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Table of Contents

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ORIGINAL ARTICLES— Page

Difficult Children, by Felix Arden 473
The Symptomatology, Clinical Course and Successful Treatment of Papuan Elapine Snake Envenomation, by C. H. Campbell and L. N. Young 478

REVIEWS—

Modern Gynæcology with Obstetrics for Nurses .. 486
Lectures on Hæmatology 486
Modern Trends in Diagnostic Radiology 486
Recent Advances in Pathology 487
Practical Dietetics 487
A Short Textbook of Midwifery 487
Staining Procedures 487
Heart Attack 487
Medical and Dental Aspects of Fluoridation 488

BOOKS RECEIVED 488

LEADING ARTICLES—

Award of the Gold Medal to Dr. H. C. Colville and Dr. W. F. Simmons 489
The Australian Scientist 489

CURRENT COMMENT—

The Anonymous Mycobacteria 490
Health Insurance 491
The Influenza Epidemic in England and Wales, 1957-1958 491

ABSTRACTS FROM MEDICAL LITERATURE—

Radiology 492
Radiotherapy 493

ON THE PERIPHERY—

On Going to Hospital 494
A City beneath a Lake 495

BRITISH MEDICAL ASSOCIATION—

Victorian Branch: Scientific 495

OUT OF THE PAST 498

OBITUARY— Page

Noel Edward Hamilton Box 498

CORRESPONDENCE—

Storage and Supply of Immunizing Material 499
Research by the General Practitioner 500
Prescription Forms 501
Hypertension and Life Assurance 501
A Case of Mistaken Diagnosis 501
Infectious Hepatitis 501
Some Factors in the Prevention of Sepsis in Ophthalmic Surgery 502

PUBLIC HEALTH—

Fourteenth World Health Assembly 502

POST-GRADUATE WORK—

The Post-Graduate Committee in Medicine in the University of Sydney 503
Surgical Seminars at St. Vincent's Hospital, Sydney 503

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 503

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS—

Primary Examination for Fellowship 504
Faculty of Anæsthetists 504

NOTES AND NEWS 504

NOMINATIONS AND ELECTIONS 504

DIARY FOR THE MONTH 504

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 504

EDITORIAL NOTICES 504

DIFFICULT CHILDREN.[1]

By FELIX ARDEN, M.D., M.R.C.P.,
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DR. HARRY SWIFT, in whose memory these lectures were founded, lived in an age when discernment in history taking and a seeing eye were even more important in diagnosis than they are today, since we supplement them with many technical procedures. But numbers of children still suffer from common conditions which cause much unhappiness, and in which the radiologist and the biochemist and the other technical wizards can offer only the limited help of excluding organic disease. In these situations we are thrown back on what we possess of powers of observation and what we have accumulated of medical experience. Children with these disorders are seldom demonstrated to students, yet they are of great importance in medical practice, and I make this my reason for discussing a few of them tonight.

The Child with Abdominal Pain.

One of the most difficult is the child who is always complaining of abdominal pain. By the time one sees him, the pain may have been present off and on for years. He has generally visited several doctors, has been offered numerous diagnoses, and has been tried on a variety of treatments with no lasting effect. Parents often remark that nobody seems to be able to get to the bottom of his case.

Such a patient was Sandra, aged 10 years, who had suffered recurring attacks of pains in the stomach since she was six years old. They had tended once to come at intervals, but now appeared almost every day, often before breakfast and again after tea. The trouble was located mainly around the navel and produced some nausea, but no vomiting. It was bad enough to cause her to miss a lot of school. Her bowels also tended to be constipated, and the stools smelt sour. Examination revealed no sign of organic disease. Sandra looked well, was exactly average weight, was not tender in the abdomen, and apart from having one or two carious teeth appeared to be a perfectly normal child. A barium-meal X-ray examination, blood count and a microscopic examination of the urine had all been done recently, with negative results. Her mother was not reassured, as I had hoped she would be, by being told that Sandra showed no evidence of organic disease, remarking quite sensibly that there must be something wrong as Sandra's pains were now worse than ever, and what was I going to do about it.

She was prefectly right, of course. There must be something wrong in cases like this, and it is our job to find out. Perhaps we should start by considering three minor ailments and dismissing them summarily as being extremely unlikely to cause pain severe enough

[1] The Harry Swift Memorial Lectures, delivered on August 25, 1960, at Adelaide.

to keep a child from school. The first of these, threadworms in the bowel, is frequently mentioned by parents; but in fact they are often symptomless, sometimes cause irritation around the anus and loss of sleep at night, and only exceptionally, when present in large numbers in the appendix, stir up some colic. The second, constipation, when severe enough to cause anything more than mild abdominal discomfort, reveals itself by hard scybalous masses in the lower part of the bowel, easily felt through the abdominal wall; but it is a commonplace to find these craggy lumps in children who have had no pain at all. The third, improperly chewed food, either from bolting meals or through lack of sufficient teeth, may occasionally give rise to intestinal colic. If there is any doubt, a short period during which all foods are well mashed before consumption can serve as a diagnostic test.

These three minor complaints did not feature in Sandra's case; but the history was revealing, and further questioning disclosed some interesting facts. Sandra was the youngest of a large family, and still in many ways the protected baby of the household. There was a distinct tendency for the pain to be worse on school days, and in fact her mother could not recall her complaining during the last holidays. It also appeared that her maternal grandmother had suffered a serious nervous breakdown a year or two previously, and an elder brother, of whom Sandra had been very fond, had died in the previous year of muscular dystrophy. Worse still, another brother was serving a prison sentence and the children at her school had found it out. Sandra had often told her mother she would like to go to a different school. She was a nice little girl, rather helpless and obviously very unhappy, and she admitted in conversation that some of the other children jeered at her because of her brother's being in gaol. I think you would agree that one would not need to drag in organic disease to explain Sandra's symptoms. In her case some discussions followed, and "Drinamyl" tablets were ordered. The rest of the story came in a letter from her mother six weeks later: "You will be pleased to hear that we removed Sandra from ——— school the very week after we visited you and she now attends ———. The results have been most satisfactory. She seems much better and altogether a happier child. She has scarcely complained of tummy-ache these last few weeks."

What do we learn from this? I am convinced that Sandra was not malingering. Very few children do; it is too difficult to carry off and too easily detected. As far as she was concerned her pains were real enough; they hurt her, and made her feel badly. But they were not due to organic disease, and must be attributed to emotional stress. Probably the actual immediate cause of the pain is increased tone, spasm or irregular contractions of the smooth muscle of the bowel wall. I think that children respond to emotional stress much more commonly with stomach-ache than with headache, as adults do, and Sandra's case is a fairly typical example.

But the story is not always the same. Peter, aged four years, was a doctor's son, and for the previous eight weeks he had been off his food and suffering attacks of abdominal pain. They tended to come after meals, which he was taking under protest. Starting rather suddenly, the pain was severe enough at times to make him cry, and had lasted as long as five hours. It returned in waves, when he would press his hands on his stomach, call out and roll around. Sometimes he would vomit, but this gave no relief. During the attacks, which happened about twice a week, he would become quite pale and clammy. Peter himself had learnt to associate the pain with meals, which he now resisted, and as a result he had lost seven pounds in weight in two months. He was not constipated and there was no contact with lead. Physical examination gave entirely negative results, a blood count and urine test gave normal findings, and the barium meal X-ray examination also revealed no abnormality, except that his appendix did not fill. The emotional history also was non-contributory. He came from an ordinary, happy home and there was no hint of stress. Laparotomy seemed indicated in his case, and it revealed a long appendix containing a fæcolith and also, stretching between the bladder and the umbilicus, a rather tight, cord-like urachus. Both this and the appendix were removed, and there were no further complaints of abdominal pain.

These two case histories, which could be multiplied with minor variations almost indefinitely, have been presented in detail to emphasize the essential difficulty encountered in almost all cases of recurring abdominal pain—that of deciding whether it is caused by organic disease or not. It is an intriguing problem when seen in this light. Too often the possibility of an emotional factor is overlooked and a differential diagnosis is attempted between different kinds of organic pains—intestinal, renal or the like—when the actual cause may in fact be unhappiness or stress.

As all of us must face this difficulty quite often, it may be helpful to outline an investigation procedure, indicating where possible points that sometimes clarify the diagnosis.

The Somatic History.

The "somatic history" is the detailed story of the pain itself, its nature, location, severity, frequency and time of appearance, together with the occurrence of any associated symptoms. In general, organic recurring abdominal pain of intestinal origin tends to be colicky in nature, and the attacks are clear-cut, with a definite starting and stopping point. They are often severe, sometimes associated with pallor, sweating or vomiting, making smaller children cry out and press their hands to their stomachs, and in most cases bringing any other activity to a standstill. They may occur at any time or be related to meals or activity, or they may wake the child at night. There is sometimes a family history of appendicitis, occasionally of ulcer. Organic pain of renal origin may be located in the loin or along the line of the ureter.

Non-organic recurring abdominal pain is more likely to be described as being present "all the time" and as being vague in relation to its period of coming and going. It is also likely to be present all over the abdomen, or in a large indefinite circle around the umbilicus. It is generally less severe, often not enough to make the child cease his play or other activity, although he may want to lie down a good deal. It seldom causes him to vomit or sweat, though it is often accompanied by headache and sick feelings. Its timing is very important. The pain hardly ever wakes the child from sleep, and it is likely to be less severe during periods of reduced emotional stress, especially school holidays and week-ends. Classically, when associated with tension at school, it starts regularly on waking from Monday to Friday and wears off during the morning. And this—I would again remind the cynics—is usually not malingering. The family history may reveal others with "nerves of the stomach" or other forms of neurosis.

The Emotional History.

When taking the "emotional history" one inquires into the family background, the school situation, friends, hobbies, interests, fears, hopes and quarrels, listening meanwhile with what has been called "the third ear" for those tones of voice that betray a mother's anxiety, over-protection, hostility or resentment and, on the child's part, whether he is happy at school and secure at home or the reverse. It is particularly important to discover, not necessarily by direct questioning, whether the abdominal pain is achieving results, in the form of parental attention and privileges or honourable absence from school, or whether it is solely a liability.

The Physical Examination.

The physical examination should be detailed and complete, and needs no comment. Investigation need not be elaborate in the ordinary way. A full blood count, microscopic examination of the urine and a barium-meal X-ray examination should be done in every case. Lead poisoning is rare nowadays even in Queensland, where it used to be common, but any hint of its possibility may be followed up by stippled-cell counts, X-ray films of the long bones and analysis of the urine for lead and coproporphyrins. Any suggestion that the pain may be of renal tract origin calls for excretion and retrograde pyelograms, and a micturating cystogram.

The Therapeutic Test.

After this, if the diagnosis is still uncertain and the pain is important enough to be troublesome, I am a great believer in the therapeutic test of a week or two in hospital. One learns a lot more about the child as a person under these conditions. Once they have accepted the doctors and nurses as friends, have discovered the new interests of play, occupational therapy, hospital television and fresh companions, and have started to forget the tensions of home and school, most children lose any symptoms they may have of emotional stress, abdominal pains among them. Organic disease, on the other hand, is unaffected, particularly if the child is allowed full activity.

Exploratory Laparotomy.

Finally, there is need in some cases for an exploratory laparotomy. It serves two purposes—the obvious one of finding and dealing with a kinked appendix, intestinal bands, intraperitoneal hernial pockets and the like, and the less obvious one of providing a most reassuring piece of evidence for the doctor who has to convince sceptical parents that their child's pains are of nervous origin only.

Treatment.

So we come at last to treatment. Organic disease, whatever the cause—and the commonest in my experience is the fæcolith in the appendix—is dealt with surgically with the appropriate combination of drama and skill. Emotionally caused pains require first that we assure the parents categorically that there is no organic disease present; and when one has convinced them of this, it is necessary to anticipate their swing to the other extreme and to assure them, with equal conviction, that the child is not "putting it on" and that the pain is a real one. Along the lines of the commonly experienced "butterflies in the stomach", we help them to realize that emotional stress does cause painful spasms of the bowel muscles, and invite them to help us find and deal with the source of the nervous tension. This may involve a change of attitude on the part of one or other parent, a talk with a school teacher, or any one of a dozen things. Anything that helps the child become less introspective, such as encouragement with friends, hobbies or other activities, is useful. No privileges, such as leave from school or special attention in the family, should be attached to having the pain, which should never be inquired after or mentioned, but there should be lavish praise and interest in any of the child's successes, handicrafts or other accomplishments. Everything possible should be done to keep him active, happy and outgoing. Some assistance may be gained in the early stages from "Merbentyl", phenobarbital or "Drinamyl". Older and more sensible children can have the mechanism of the pain explained to them, with the assurance that under the new pattern of living the contractions of the bowel muscle will not break through into consciousness much longer, and they will gradually find the pains disappearing altogether.

Children with Sick Feelings.

Very similar in many ways to children with abdominal pains, and equally difficult to deal with, are those who regularly complain of feeling "sick in the stomach". As before, we are confronted with a boy or girl, usually in the latter half of childhood, and often of sensitive disposition, in whom we are unable to discover any abnormal signs by the most careful examination. Yet the symptoms sound genuine enough, and the episodes of sickness—either a state of feeling sick or actual vomiting—are interfering with the child's activities and happiness. As before, we have the difficulty of making a diagnosis and of determining whether the sick feelings are caused by emotional stress or some organic disease. I want to suggest that it is possible to do this in many cases on the history alone if we ask enough of the right questions. Here is the story of a child whose symptoms were of psychogenic origin.

Judith, aged 11 years, had been feeling "sick in the stomach" for about five months when I first saw her. She was usually well in the evenings, slept well, and woke in the morning feeling as if she was going to vomit, although she never actually did so. With the sick feeling she had a certain amount of upper abdominal discomfort, and she was forced to lie down for the early part of the morning. The symptoms tended to wear off during the day. Her parents were concerned because she was missing a great deal of school and her education was being affected. That was the story as related by her mother, and it might have meant anything; but it took on more significance when certain gaps were filled in. The sick feelings, it seemed, were not enough to take away her appetite for breakfast, which at times included porridge and bacon and egg. They had started about a week after the commencement of the first school term, and were much less noticeable over the week-ends and during the holidays. Judith was the youngest of three children, but virtually an only child, as the others were grown up and married. She had practically no outside activities, lived rather far away from the few friends she possessed, and was not allowed a bicycle. She did not belong to any clubs or organizations. My notes describe her as a tall, slim, pale, sad-looking and introspective girl who had already reached the age of puberty. She was not anæmic, and physical examination revealed no abnormality. I am sure you would agree that she required hobbies, companions and other interests, and that some stress probably needed settling at school. It proved to be a young aggressive male teacher of whom she was afraid. Judith has now joined the Girl Guides and feels happier, and her mother has been to the schoolmaster to ease the situation there. Judith has also started dancing lessons, and has learnt that sick feelings are only a symptom of slight nervousness, common enough in girls of her age, and that her tablets will take them away, and she is beginning to take a pride in being able to disregard them. Her mother now describes her as much happier and very well.

For contrast, may I tell you another story?

This concerns Lyndall, another girl, aged eight and a half years, who for some time has been getting attacks of nausea, vomiting and other symptoms at intervals of about four weeks. All the attacks are very similar to one another. Having gone to bed quite well the previous night, she wakes early in the morning feeling sick and she vomits soon afterwards. She usually has also some mild abdominal pain, a slight rise in temperature and frontal headache. The nausea and vomiting generally last about 24 hours and the headache about 48 hours, but she is so washed out afterwards that her mother usually keeps her in bed for a week and away from school for another week while she picks up. Lyndall, I may add, is an only child. During the first day of each attack she cannot look at food, and she is pale and has a bad breath. Various diagnoses have been offered, including "sinus trouble", "hepatitis without jaundice", and "a virus disease that is going about", but her mother, a most intelligent person, commented that, if this was the case, "she wouldn't keep on getting it, would she?". Questioning revealed that both Lyndall's grandmother and great-grandmother suffered from migraine, and that her own mother periodically experiences right frontal headache which lasts as long as two days. This suggests strongly that Lyndall's troubles are also a juvenile form of migraine.

I am sorry that there is not time for a full discussion of juvenile migraine this evening, for I believe it to be a relatively common disease which does not receive the attention it deserves, either in textbooks or from many practising doctors. It is almost certainly the correct designation for what is called cyclical vomiting, acidosis or bilious attacks. Such children commonly have a family history of migraine, and if watched until adolescence can often be observed to lose the vomiting component of their illness and to develop instead the recurrent headaches of the grown-up. Many an adult migraine sufferer can remember the bilious attacks of his early childhood. All this is fairly well known and accepted, but it does not seem to be so commonly realized that episodic illness such as Lyndall's is also likely to be juvenile migraine. In these episodes, which tend to repeat one another fairly accurately, and often at fairly regular intervals, the child suffers from any or all of the following—nausea, vomiting, abdominal pain, headache, fever, anorexia,

pallor, constipation and ketonuria—sometimes one symptom predominating and sometimes another.

The important distinction between this and the more indefinite sick feelings of emotional origin is that juvenile migraine comes in attacks or episodes, the child being quite well and not in the least unhappy between whiles. It is essential to make this distinction, because a correct diagnosis of migraine indicates that the child is not suffering primarily from stress, although his attacks may be more frequent then, and is not likely to be cured either by psychotherapy or by readjustment of his background. On the other hand, it does enable one to tell the parents that the trouble is more uncomfortable than serious, and that although the attacks cannot be prevented, they may to some extent be relieved. "Largactil" and related drugs seem to be best for the nausea and vomiting, and ergotamine preparations when headache predominates.

I am not suggesting that all children with sick feelings can be included in these two categories, eager though I am to secure proper recognition for the concept of sickness from emotional stress, and for juvenile migraine.

I saw recently an intelligent little girl of six years, the only child born to her parents in 11 years of marriage. They were cultured and sensitive people and completely devoted to her. She had grown up in their company and had few outside friends, and in consequence she had been unhappy at school, particularly as they had moved to a new town a short time before. For some while she had complained of sick feelings and general tiredness, and was described as being cranky and restless at night. During the previous 10 days the feelings of nausea had grown worse. She had vomited one evening, and every morning since, missing school and indeed being in bed for the past five days, as her mother believed she was not well. However, apart from feeling sick, the child had no complaints and, in particular, no headache. On the history and family background alone, one might have felt little anxiety; but on examination this child exhibited right facial weakness, cerebellar ataxia and incoordination. Subsequent investigation revealed an inoperable brain-stem tumour.

In a sentence, the only generalization one can safely make about organic disease as a cause of sick feelings is that the history is usually short and that there are likely to be accompanying physical signs.

Fat Children.

Though much happier and more likeable as patients than the previously mentioned two groups, fat children also present difficulties from the point of view of management. In this abundant land it is common enough to see youngsters who are moderately overweight, and we surely will so long as parents feel it their duty to introduce as much nourishment as possible. I am not concerned with them tonight, but with those grossly obese children who, because of their size, have become a source of real discomfort to themselves and an embarrassment to their parents.

One such difficulty happened recently. In sailed mother, herself enormous, bringing Denise who, at three years and two months, weighed 72 lb., compared with the average of 33 lb. for her age. When she had regained her breath, the mother said that she herself had suffered from "gland trouble" all her life and did not want a repetition in the child. She carried a letter from a doctor suggesting Fröhlich's syndrome. One learned that Denise was a big baby, had always been in excellent health, did not eat much and was very active. There were several other heavy people on both sides of the family. Examination showed Denise to be a cheerful, intelligent child, large in all directions (she was four inches over average height as well), with a florid face, knock-knees, somewhat Cushingoid appearance and slight elevation of the blood pressure. An X-ray picture of the pituitary fossa was normal.

Perhaps we should start discussing fat children at the level of the complaint that it was "gland trouble", and ask ourselves to what extent gladular dysfunction is responsible for obesity. Belief in this relationship is widespread, and it has probably gained adherents lately, with the evidence of the fat cheeks, large appetite and general obesity of some children on long courses of certain steroids.

People tend to be uncertain about which gland they believe to be at fault. Fröhlich's syndrome, as originally described, was a combination of obesity, hypogenitalism and pituitary cyst or tumour, and should not be diagnosed without X-ray evidence of enlargement of the pituitary fossa. Fat boys may show an illusion of hypogenitalism by having a mound of fat in the pubic region which almost conceals the penis. This is confusing, but it can be shown by pressing the fat away with the hand that the organ is of normal size. I cannot recollect seeing a genuine case of Fröhlich's syndrome, which must be quite uncommon.

To my mind there is some truth—but not a great deal—in the glandular theory of obesity. This is based on evidence of excess growth hormone from the anterior pituitary lobe (some of these children being large in their skeletal structure as well as being fat, having tall relatives and starting life as large babies), and also on evidence of excess adrenal cortical activity (some of them showing the plethora, striæ on the buttocks, slight hypertension and general vague similarity in appearance to children with hypercortisonism).

On its own account, no kind of glandular dysfunction can cause a child to put on weight. Hormones can increase the appetite, or facilitate food assimilation or reduce its metabolic combustion. They probably alter as well the little-understood safety-valve mechanism which normal people seem to possess, so that excess food, instead of being burnt off and breathed out, as usually happens, is converted into fat and stored in the depots for the rainy day that never comes.

It is time we stopped thinking and saying that fat children are suffering from "gland trouble", because this conveys a sense of irresponsibility and hopelessness about treatment. They are really suffering from "primary obesity" due to the basic fact that their food intake is in excess of their actual needs. In many cases this is an inherited characteristic, and it is often assisted by a family pattern of over-eating. Sometimes the intake is not abnormally high; but it must be, by the simple arithmetic of calorie needs and caloric consumption, too high for that particular child, as otherwise he could not be gaining weight. Some animal experiments may be quoted. Rats, it is known, become extremely fat after injury to the hypothalamus, and this is taken as evidence for glandular dysfunction; but the increase is not due to any mysterious effect on body metabolism, but simply to appetite stimulation. When paired animals are used, one being kept as a control, hypothalamic damage causes no weight gain so long as both eat identical measured diets. But when both are allowed free access to food, the hypothalamic rat, with his voracious appetite, rapidly increases in weight relative to the normal one.

I am certain that the only way to help fat children is to control their food intake. We can do nothing about their excellent digestions and unusual fat-storage mechanisms, and attempts to increase food combustion by ordering exercise or prescribing thyroid seem only to improve their appetites. Many of these youngsters are surprisingly energetic. A recent patient of mine was, unknown for a time to his mother, walking two miles each way to school so that he could use his bus fares to buy sweets. Can these children, then, be usefully treated at all?

To present a practical answer one might describe the case of a girl, aged 13 years, who was supposed to suffer from thyroid trouble, for which she took three grains of thyroid daily. She had always been huge and was said to have weighed 44 lb. at 14 months. Now she scaled 158 lb., although she was 3·5 in. below normal height, the average for girls of her age and stature being only 80 lb. Her appetite was tremendous, and she would help herself all day from the refrigerator if she was not controlled. It made her mother's task more difficult that she was somewhat backward and attended an opportunity school.

The management of such patients is on the following lines.

1. Both mother and child should be told that one understands the problem and can help them. The child should be coaxed into wanting to lose weight and into being prepared to try hard to keep to a diet.

2. An explanation should be given about the causation of obesity and of how weight loss cannot fail to occur if the food intake is below total requirements.

3. A diet sheet should be supplied which, briefly, eliminates milk as a beverage (allowing skimmed milk for cooking) and makes heavy cuts in sugar, cereals and fat, while allowing proteins, fruits and vegetables in unlimited amounts.

4. Drugs to reduce appetite are prescribed. (If primary obesity is an over-eating disease of hypothalamic origin, as it seems to be, the excess appetite needs to be attacked at source. Young children will not reduce their intake either to please mother or through disciplinary methods, though when they are old enough to be teased and sensitive about it they may exert remarkable self-restraint.) Useful drugs are methyl amphetamine (5 mg. on rising, with perhaps another 2·5 mg. at midday), or "Tenuate" (one tablet three times a day, an hour before meals).

5. "Bulk satisfiers" are suggested, which include not only unlimited fruits and vegetables, but also methyl cellulose products which can be obtained as biscuits. One or two of these with water, unsweetened orange juice, beef-tea or weak black tea, relieve the empty feeling between meals. Synthetic sweetening agents can be used extensively instead of sugar, and chewing gum in place of other sweets.

6. Regular weighing and encouragement are psychological necessities. Good resolutions falter and food is very tempting. A record of weights, weekly at first, then monthly, keeps both child and parent aware of progress, demonstrating now the need to try harder, now a permissible easing.

Finally, what sort of results can one expect? Denise, who recently started treatment on "Tenuate", lost 7 lb. in the first month. Margaret, the backward girl, takes methylamphetamine and is still big, but is now only 21 lb. above average for her age and height, whereas in 1958 she was 78 lb. overweight. Without tablets her appetite is still colossal and (in her mother's words), "if left alone she'll eat and eat and eat for ever. Her grandfather is just the same and he's real fat too".

The Subnormal Child.

Let us assume tonight that we are seeing for the first time a child of about six years, brought because he is "lazy" and not concentrating at school and therefore doing badly; and also perhaps because he is speaking poorly, clumsy in his behaviour and prone to tantrums. It is quite likely that we will detect a note of blame for the child in the report that is being given about him, for the idea that he may be subnormal has not even occurred to his parents, and the teacher perhaps regards his poor performance as lack of effort when it is really a lack of ability.

A child like this poses all sorts of difficult problems. We have to determine whether he is indeed backward, or alternatively of average intelligence, but failing because of some emotional stress at home or at school. If he is subnormal, we have to discover the reason for it, if possible. The degree of his mental defectiveness needs to be determined accurately, and any physical disabilities located so that they may be corrected. And when we know as much as can be learnt about the child and his capabilities, mental, physical and emotional, and about his home and family background, we must attempt to forecast his future potential and advise his parents about his training and management.

I am not suggesting that any of us are capable of doing all this single-handed. We shall certainly need the assistance of a child psychologist and often of a social worker; but we are the people to whom the parents first come for assistance, and we have to provide the continuing guidance and advice.

Ascertaining whether the child is backward or not involves going back to first principles, with a detailed history of behaviour and performance from the time of birth onwards. Did he, for example, have to be put into oxygen when first born? Was the doctor worried about him in any way? Was he able to be brought to his mother on the first day, and was he able to suck at once? With these and many other questions one patiently takes his mother through the developmental milestones of infancy. On coming into childhood, there are certain other stages of development which the average child passes in succeeding years, but it must be remembered that wide variations occur. It helps a lot if there is a younger sibling, cousin or close friend with whom to compare the patient, and one may discover, for example, that at six he talks less clearly and with a smaller vocabulary than his four-year-old brother, and seems less interested in picture-books and stories.

Clinical examination is more for the purpose of finding physical defects than the level of intellectual attainment, but it must be thorough. One looks, of course, for signs of hypothyroidism, though I find this an uncommon condition. Frank cretinism and phenylketonuria are not encountered in this situation, as such children, at six, are gravely defective. I should mention serological tests for syphilis, but admit having abandoned them myself unless there are special indications. However, deafness is extremely important. Some children are labelled stupid when they are really deaf, not totally, but enough for them to miss a good deal of what people are saying. Visual capacity also needs careful testing. Lesser degrees of spasticity, also, may be responsible for apparent clumsiness, just as muscle hypotonia may have caused delay in walking. Speech defects may be evident, but are rarely if ever due to tongue-tie. Periods of lack of attention may in fact be episodes of minor epilepsy, and it is well to exclude these, if there is the slightest doubt, by an electroencephalogram.

While the examination is going on, one notices, if present, the physical stigmata that may accompany mental defect, particularly those of the head, ears and fingers.

Examined recently was Jenny, who was seven years old, referred because of poor progress at school. Her parents had started her the previous year, but after three months the teacher had asked that she be removed and she had gone to a kindergarten. This year she was doing no better, her progress being described as "hopeless". She would not concentrate, spoke poorly and was clumsy and always tripping over. She had once fallen out of a bus on the way to school. Her parents thought she did not see well and wondered about her hearing, as she had suffered from otitis media. She was hard to discipline at home and had tantrums. She had passed her baby milestones at the usual times, except that she had not talked until she was four years old. A younger sister, aged five and a half years, excelled her in every way, and even the youngest, aged three and a half, spoke more clearly. Jenny could dress and feed herself and knew some nursery rhymes, though her speech was very poor. Her head was slightly smaller than average, and although she was not mongoloid, her little fingers were short and somewhat curved. Her mental age proved to be only three and a half years, which was of course the reason for her failure at school.

One may make a reasonable guess at the mental age of a child in one's consulting room; but its accurate assessment demands careful testing by an experienced person and is always a matter for an expert. It is a valuable piece of information for anyone proposing to offer guidance on a subnormal child.

Let us say that we have decided that our patient is backward, and with expert assistance have reached an estimate of his approximate degree of backwardness. His parents will naturally want to know what has caused the trouble, and we need to have in mind some sort of classification of the principal ætiological factors. They may be briefly set out in the following table:

Pre-Natal Causes:

Infections (toxoplasma, syphilis, rubella).

Hereditary causes (cranial anomalies, phenylketonuria, others).

Maternal irradiation.

Cretinism.

Unknown causes (mongolism, cerebral palsy, primary amentia).

Natal Causes:

Birth trauma, mainly anoxia.

Post-Natal Causes:

Kernicterus.

Degenerative cerebral diseases.

Infections (encephalitis, meningitis).

Head injury.

Lead and other poisons.

As a rule, the history and examination reveal the cause readily enough when one exists. But the majority of cases are due to developmental defect of the brain for no reason that we can yet understand, and it is therefore important, once again, not to let the parents blame themselves or anyone else for this all too common and seemingly natural event.

Having accepted the fact that their child is backward they will possibly next inquire whether he will have to be sent to a home. This question brings up the whole matter of future management, and the answer depends on the child's actual status. Let us consider three groups, and agree that children of less than mongoloid capacity—or, to put it another way, children whose mental age proves to be less than half their actual age—are best admitted to an institution at once for permanent care.

At the other end of the scale, children above mongoloid capacity are generally happiest in their own homes, provided that the family is a united one and living conditions are normal. They need love and understanding, of course, and in particular they need praise for their daily achievements, however simple. Too often we forget that backward children, as they grow older, are sensitive, and well aware of their lack of skill and success in nearly all the things that make up daily life. Training them for simple routine household jobs, such as laying a table, watering the garden or feeding the chickens, and frequently telling them how useful and helpful they are becoming, and how good it is to be able to rely on them, not only sweetens family life but makes these handicapped youngsters feel important, and gives them a sense of fitting into the only community they understand. Everything that we doctors can say and do to help parents accept these children as affectionate little helpers, and not as liabilities, is worth while. Everything that helps them develop independence and responsibility should be tried. There are all sorts of simple handicrafts that they can attempt. Older boys can grow vegetables in the garden, sell them to mother for pennies and spend the pennies in the shop. Girls can help with shopping and housework. They can watch for the postman and bring in the letters, and so on. And every day their parents ought to be able to find something for which to praise them. Discipline is necessary, of course. There must be rules in every home, and these must be kept; but scorn and criticism simply wilt them. Jenny, the little girl I mentioned earlier, was thawing nicely after a shy start, and had just whispered confidentially "I got a sore tooth, too", when her mother cut in coldly: "He's not a dentist, stupid." Jenny was seven, you may remember, and her mental age was three and a half. As childhood ends, there come the problems of fitting these children for adult life. Unfortunately I have no time to discuss this, except to say that the rehabilitation programme of the Commonwealth Social Services Department has plans for training the intellectually, as well as merely the physically, handicapped. In some parts of the world progress has been made in teaching simple routine manual tasks to subnormal adolescents, even down to the imbecile level.

So much for those below and those above mongoloid level. The third group involves mongoloid children themselves. Authorities have differed about their management, some urging that they be brought up at home and others that they be separated from their parents at birth and sent to an institution. My own views about them are between the two. There does certainly come a time, around the age of eight years, when their mental age is about four years, at which they need the companionship of others of their own kind and seem happiest in groups. From this stage onwards institutional care is right and proper. But in infancy they are like any other babies, save that they remain so for a longer time, and their needs are identical, and in early childhood they need the individual care and affection that only a mother can give, and definitely develop faster and further than those unfortunate enough to be in State homes.

I promised to tell you more about a child whose parents had been informed at her birth that she was mongoloid and that there was "no hope" for her. Just the other day a letter arrived, and I read:

> The dearest and most adorable child that ever gladdened a parent's heart died on June 17th. . . . Really, Doctor, she was as sunshine in a dark room and we all so dearly loved her. . . . At present our loss seems irreparable but we will always remember the love that she brought to our house.

It adds point to this letter to explain that it was written, not by the mother, but by the father, and that he was a husky member of a State police force.

Conclusion.

There is enormous scope, of course, in pædiatric illnesses, for medical science in all its aspects. But there is also a place, where children are concerned, for more than mere science, for children are complex little beings, and technical skill alone can never discover all their secrets. From the reports of those who knew him best, it was this other special quality, an understanding heart, that Harry Swift possessed. It was one of his greatest attributes, one of the factors in his success.

THE SYMPTOMATOLOGY, CLINICAL COURSE AND SUCCESSFUL TREATMENT OF PAPUAN ELAPINE SNAKE ENVENOMATION.

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The Papuan and Australian venomous terrestrial snakes all belong to the family Elapidæ. The most venomous Papuan species, the taipan and the death adder, are common to both countries, while a third highly venomous and common Papuan snake, the Papuan black snake, is related to some common Australian venomous snakes (the red-bellied black snake and the king brown (mulga) snake).

Elapine snake venoms are complex mixtures of proteins with enzymic activities (Braganca and Quastel, 1953), and usually have their main toxic effects on the nervous system. After a serious elapine snake bite in man a characteristic clinical picture is produced, the main feature of which is a progressive paralysis of all the ocular, bulbar, jaw, facial and spinal muscles, with eventual death from respiratory obstruction or terminal peripheral failure of the diaphragm.

It has long been known that this paralysis, however severe it may be, is completely reversible if a "sublethal" dose of venom has been received, and it has always

been the hope of workers in this field that the paralysis would still prove to be completely reversible even after the injection of potentially lethal doses of venom, if the patient's life could be maintained during his period of respiratory insufficiency. Up till now there has been little, if any, clinical or experimental work to justify this hope.

We wish to record here our observations of 15 cases of elapine snake envenomation of varying severity, and to report successful treatment in potentially fatal cases of severely paralyzed patients, with antivenenes, tracheotomy and artificial respiration.

The patients were all Papuan natives admitted to the Port Moresby General Hospital over a ten-month period from November, 1959, to August, 1960, and were brought to hospital by road or aeroplane from within one hundred miles of Port Moresby. As many of the patients were too ill to give a history on admission (some being unable to speak), most of the histories were obtained through interpreters, retrospectively, after the patient recovered. As a consequence of this, the subjective sensations resulting from snake envenomation may be inadequately described. After the patient's admission to hospital, an hourly record of the pulse and blood pressure was kept throughout the first few days of the illness, and the progress of the condition was closely observed.

The main features of each case are summarized in Table I, and the clinical picture and treatment are described in more detail below.

Circumstances of the Bite.

With the exception of one, all the patients were bitten in the daytime. Each was barefooted at the time of the bite and was bitten on the foot or the leg. The bite occurred while the patient was walking through long, coarse grass, from one to several feet high, or along a bush track, from one to three feet wide, bordered by this grass. Three patients actually trod upon the snake. The other patients did not see the snake before it struck, and some never saw it at all. Only three patients thought that the snake grasped the bitten part between its jaws.

Clinical Picture.

Local Symptoms and Signs.

In the majority of cases there were no local symptoms or signs of any significance. Pain at the site of the bite was usually absent or mild. Severe pain in the whole of the bitten limb was complained of by only one patient. This man also had tender, enlarged inguinal lymph nodes, which were noticed within 12 hours of the bite. Slight œdema of the bitten part was present in four cases. A fifth patient, who had been bitten on the leg, had extensive swelling and œdema of the leg and foot, which persisted for 11 days. (In this case a tourniquet had been left on for four hours.)

General Symptoms.

Some symptoms or signs of systemic envenomation were always present within 12 hours of the bite, and often the first symptoms appeared within two or three hours. Vomiting was one of the earliest symptoms, the patients usually vomiting several times. Three patients also vomited small amounts of blood. After the vomiting ceased, three patients developed abdominal pain which lasted a few hours. (We have seen a patient suffering from severe snake bite admitted to hospital with a provisional diagnosis of acute appendicitis, owing to complaints of vomiting and abdominal pain associated with marked abdominal tenderness.) Six patients complained of mild headache, which lasted from a few hours to two days.

In addition, the patients complained of one or some of the following symptoms: drowsiness, blurred vision, double vision, inability to open the eyes, difficulty in speaking or swallowing or opening the mouth, general muscle weakness, heaviness of the bitten limb and the passing of blood-stained urine.

Table I.

Summary of the Clinical Features, Treatment and Results of 15 Cases of Elapine Snake Envenomation.

Case Number.	Approximate Age. (Years.)	Sex.	Symptoms and Signs.															Treatment.										Recovery Period. (Days.)	Residual Disability or Outcome.
			Paralysis[a] of															Antivenene.[3]											
			Headache.	Vomiting.	Abdominal Pain.	Visual Disturbance.	Eyelids.	Eye Movement.	Face Muscles.	Tongue.	Palate.	Jaw Muscles.	Head, Neck.	Intercostals.	Diaphragm.	Arms.	Legs.	Bleeding Tendency.	1/M.[4]			1/V.[5]			Tracheotomy.	Artificial Respiration.	Prognosis without Treatment.		
																			Tiger.	Taipan.	Papuan Black.	Death Adder.	Tiger.	Taipan	Papuan Black.[6]	Death Adder.				
I	17	M.	?	Yes.	No.	Yes.	+	++	++	++	++	+	+	++	+	+	+	+	6	6							Yes.	Yes.	8	None.
II	20	M.	?	Yes.	No.	Yes.	++++	+	+	++	++	+	++	++	+	+	+	+					18	24			Yes.	Yes.	5	None.
III	35	M.	?	Yes.	No.	?	+	+	+	+	+	+	+	+	+	+	+	+					18	24			No.	No.	21	None.
IV	25	M.	No.	No.	No.	No.	++++	+	+	+	+	+	+	+	+	+	+	+					30	30			No.	No.	9	None.
V	10	M.	No.	No.	No.	No.	+	+	+	+	+	+	+	+	+	+	+	+					12	36			No.	No.	—	Death.
VI	30	F.	Yes.	Yes.	Yes.	Yes.	++	++	+	++	+	+	+	+	+	+	+	+	24	3			24	18			Yes.	Yes.	5	None.
VII	35	F.	No.	No.	Yes.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+					30	30			No.	No.	6	None.
VIII	13	F.	No.	No.	No.	No.	+	+	+	+	+	+	+	+	+	+	+	+					24	24			Yes.	Yes.	5	None.
IX	25	M.	No.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	6	3			30	27			No.	No.	5	None.
X	14	M.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+					18	30			No.	No.	1	None.
XI	40	M.	Yes.	No.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+		3				30			No.	No.	8	None.
XII	20	M.	Yes.	No.	Yes.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	9	3				30			No.	Yes.	62	Yes.
XIII	25	F.	?	Yes.	?	?	+	+	+	+	+	+	+	+	+	+	+	+	15	9			45	30			Yes.	No.	+	None.
XIV	17	M.	No.	No.	Yes.	No.	+	+	+	+	+	+	+	+	+	+	+	+				30			30		No.	Yes.	6	None.
XV	25	M.	Yes.	Yes.	No.	Yes.	+	+	+	+	+	+	+	+	+	+	+	+	6			36	30	36			Yes.	No.	21	None.

[a] "–" No deviation from normal. "+" Severe degree of involvement. [3] Thousands of units.
[4] I/M=intramuscular administration. [5] I/V=intravenous administration.

General Signs.

Nervous System.

Bilateral ptosis was present in all cases. The paralysis of the eyelids was partial or complete, depending on the severity of the case, and was usually the earliest objective sign of snake envenomation to develop, appearing as early as two hours after the bite. This was followed by impaired ocular movements in a vertical or lateral direction. The ocular movements gradually became more restricted in the serious cases until no movements were detected at all. The eyes were fixed and central. The complete ptosis with the fixed central eyes presented quite a charactertistic clinical picture. The pupils were slightly to moderately dilated and always reacted to light.

In all but the mildest cases, the muscles of the jaw, the palate and the tongue were involved, usually after paralysis of the eyelids and the extrinsic eye muscles had commenced. The degree of their involvement varied from slight weakness, which was manifested by a nasal voice, slight difficulty in swallowing and inability to open the mouth completely or to protrude the tongue properly, to complete paralysis. In the last-mentioned circumstance, the patients were unable to speak or swallow at all, and secretions accumulated in the pharynx. The tongue was completely immobile on the floor of the mouth, and no movement of the palate could be detected. The patients were unable to open or close the mouth, the teeth being only a quarter of an inch apart. Sometimes the jaw was freely movable backwards and forwards by hand, and in the supine position, with gravity, it readily fell backwards, leading to respiratory obstruction. The patients with the most severe paralysis also had weakness or almost complete paralysis of all the muscles of the face. They were unable to wrinkle the forehead or move the lips or nose significantly. Complete paralysis of the muscles of the face developed late in the course of the illness.

A variable degree of involvement of the muscles of the neck, the trunk and the limbs was present in all but two cases. Usually the neck and trunk muscles were more extensively paralysed than the limb muscles. Often the patients were unable to move head or trunk at all. The limbs were symmetrically paralysed in nine cases, the proximal muscle groups always being more affected than the distal ones, and the paralysis was never complete. Even in the most severe cases, a finger or a hand, and some toes or a foot, could still be moved slightly. In three cases the muscle weakness of the limbs was more marked in the bitten limb.

Paralysis of the intercostal muscles usually occurred only after there was extensive paralysis of all the ocular and bulbar muscles. In the most severe cases the paralysis was complete. Respiration became entirely diaphragmatic in character. Progressive weakness of the diaphragm then occurred over a period of many hours, eventually terminating in complete failure of this muscle. Difficulty in breathing was thus a feature of all the serious cases, and was due to respiratory obstruction, or to paralysis of the intercostal muscles and the diaphragm. Observation of other patients indicates that death finally occurs from the effects of anoxia resulting from failure of the diaphragm, from respiratory obstruction or a combination of both these factors.

All the patients appeared drowsy on their admission to hospital, and some were cyanosed and occasionally restless. All but one patient remained cooperative in spite of severe paralysis, and appeared to hear and tried to obey commands. However, on recovery two of the most severely paralysed men (Cases I and XV) could not remember any details of the earlier part of their illness. One of these men (Case I) had had some injections of paraldehyde. One patient (Case XIII) appeared confused and restless throughout the earlier part of his illness, but this was atypical.

In five of our less serious cases, no abnormality in the deep tendon reflexes was detected. In the other cases, depending upon the severity of the limb paresis, there was a decreased reflex response or a complete absence of deep reflexes for some days.

Whenever possible, sensation (pain, light touch and, occasionally, vibration and joint sense) was tested. No generalized sensory changes were detected. In three patients there was some loss of pain and touch sensation over a small circumscribed area in the vicinity of the wound.

In cases in which muscle weakness alllowed it, muscle coordination was tested and no impairment was found. There was no disturbance of micturition.

Cardio-Vascular System.

There was singularly little involvement of the cardiovascular system. The blood pressure was usually well maintained (any significant alteration being related to inefficient ventilation of the patient or excessive bleeding from the tracheotomy wound, and responded quickly to treatment of the underlying cause). Transient tachycardia was observed in several cases before tracheotomy was performed, but once the anoxia was relieved the pulse rate returned to normal.

Urine.

Eleven patients had albuminuria, which persisted for three to four days. However, in some cases, the urine was obtained after the serum had been given. The centrifuged deposit of the urine sometimes contained up to 10 red cells or polymorphonuclear leucocytes per high-power field, with granular and hyaline casts occasionally being noted. One patient (Case V) passed heavily blood-stained urine after admission to hospital and another man (Case XIV) passed black urine. In these two cases these signs were the first objective evidence of envenomation, and they were followed by mild neurological signs.

Blood.

Some patients bled freely from the first-aid incisions and later from the tracheotomy wound, for one to three days. Venepuncture wounds in these patients were also noted to bleed excessively. Because of the presence of anoxia and recent first-aid trauma, together with the fact that blood counts were sometimes not obtained until after the serum had been given, no significance could be attached to changes in the white-cell count.

Cerebro-Spinal Fluid.

In two cases lumbar puncture was performed. The pressure was normal, and no abnormality was detected on microscopic or biochemical examination of the fluid.

Treatment.

First-aid treatment had been attempted in three-quarters of the cases. All the patients had been bitten many hours before admission to hospital. The shortest period between the bite and admission was four hours. The most urgent need on admission was the establishment of an adequate airway. A tracheotomy was performed under local anæsthesia in nine cases as soon as possible after admission. A cuffed tracheotomy tube (James tube) was inserted. Lives were sometimes saved by passing a cuffed endotracheal tube in the ward. Because of a shortage of trained staff throughout the 24 hours, we have felt it safer to err on the side of caution in performing an unnecessary tracheotomy, rather than have the patient develop obstructed breathing at night. The tracheotomy tube was usually left in for four to seven days. An anæsthetic machine (utilizing a semi-closed circuit with a soda-lime absorption unit) was used manually, mainly by semi-trained staff, to assist the patient's failing respiration in four cases. The longest period over which respiration had to be assisted was 84 hours.

When there was difficulty in swallowing, oral feeding was suspended and hydration was maintained by intravenous infusions of isotonic glucose-saline solutions. This usually had to be continued for three to seven days. Two patients, who bled freely from tracheotomy wounds, had to have blood transfusions.

The oral and tracheal secretions were sucked out as required. The patient's position was changed and the cuff of the tracheotomy tube was deflated every two hours. As a prophylactic measure antibiotics were given intramuscularly, and if required, intramuscular injections of paraldehyde were used to sedate the patient.

The Commonwealth Serum Laboratories have prepared three specific antivenenes—namely, taipan, death adder and Papuan black snake—for use in Papua. Australian tiger snake antivenene is also freely available, and is said to give some cross-protection in cases of bites by other venomous snakes. Antivenenes were given to all patients in quite large single doses intravenously (up to a total of 90,000 units). Some of the patients had already received small or large doses of antivenene (usually tiger snake) prior to admission to hospital.

Usually two or three types of antivenene were given to each patient in doses of 12,000 to 30,000 units each. If the snake was seen and it was a small one, death adder antivenene was included in the régime. If excessive bleeding was present, Papuan black snake antivenene was also given, but otherwise no set régime has been followed. Most commonly, taipan and tiger snake antivenenes were given together.

Untoward reactions to the serum were usually not serious; pyrexia, rigors, periorbital œdema, wheezing, coughing and pruritus were encountered. One patient died from acute pulmonary œdema, and it is thought that this was probably due to a reaction to the antivenene or to the excessive intravenous administration of fluids (Case V). Several injections of negostimine were given to two patients with severe paralysis without any beneficial effect.

Stage of Recovery.

In the mild cases, quick and complete recovery occurred, often within two to three days of the bite. In the most severe cases, one to three days after the bite the diaphragm movements became stronger and did not need assistance. The faintest flicker of eye movement was seen then or shortly before this (usually two to two and a half days after the bite), and within the next 12 to 24 hours definite eye movements were detectable. Completely normal eye movements were present within the following 24 hours. At the same time the ptosis decreased. The paralysed bulbar muscles improved more slowly, but the patient was able to swallow and speak within four to eight days of the bite. The weakness of the limb muscles diminished before that of the neck and trunk muscles. A complete recovery had usually occurred within five to 14 days of the bite. However, in one case the bulbar paralysis has persisted for six to ten weeks and improvement is still occurring. This patient has residual disability also from an upper motor neuron lesion affecting the left side of the face and the left hand (Case XIII).

DISCUSSION.

Species of Snake Inflicting the Bite.

Five venomous terrestrial snakes in Papua are capable of inflicting a fatal bite (Slater, 1956)— namely (i) the Papuan taipan (*Oxyuranus scutellatus canni* Slater); (ii) the Papuan black snake (*Pseudechis papuanus* Peters and Doria); (iii) the death adder (*Acanthophis antarcticus rugosus* Loveridge); (iv) the Papuan whip snake (*Demansia psammophis papuensis* Macleay); and (v) the small-eyed snake (*Micropechis ikaheka fasciatus* Fischer). As bites from only the rare, very large specimens of the last two species are likely to be lethal for man, fatal snake bite in Papua is due most commonly to a bite from a Papuan taipan, a Papuan black snake or a death adder. These three reptiles are all highly venomous and are not uncommonly encountered.

For its size (usually two and a half feet or less in length), the Australian death adder was regarded by Fairley (1929*c*) as the world's most deadly terrestrial snake. However, the taipan, because of its large size (up to 11 feet in length), its efficient biting apparatus and its production of a venom which is only slightly less toxic than that of the extremely potent Australian tiger snake venom (Morgan, 1956), is generally accepted as Australian's most dangerous snake (Kellaway and Williams, 1929; Morgan, 1956). The venoms of the Papuan taipan and the Papuan death adder are possibly very slightly more toxic than those of their Australian counterparts (Slater, 1960). Papuan black snake venom is much more toxic than the Australian black snake venom, but although this large Papuan snake has a high venom yield, the venom has only about one-eighth the potency of taipan venom (Slater, 1960). The taipan and the Papuan black snake are active by day, while the death adder is active by night (Kinghorn, 1956; Slater, 1956), and was regarded by Fairley (1929*c*) as the only Australian venomous snake to be encountered commonly at night.

In none of the cases reported above was the species of the snake causing the bite determined. This is a common experience of those dealing with snake bite clinically (Acton and Knowles, 1914; Clark, 1942; Ahuja and Singh, 1954), and raises considerable difficulties in connexion with antivenene treatment.

Factors Determining a Lethal Bite.

The presence or absence of symptoms following venomous snake-bite, and the rapidity with which symptoms develop and lead to death, are mainly dependent upon the amount of venom injected in relation to the size of the victim. For a given venom, the death time (the interval between the bite and death) is roughly proportional to the amount of venom injected (Acton and Knowles, 1914).

Apart from its size, many factors in the ecology of the snake, such as moulting, disease, recent feeding or biting and hibernation, influence the amount of venom available for injection at a given time. Individual variations in the venom yield also occur from time to time in the same snake for no apparent reason (Fairley, 1929*c*). Inefficient biting also may considerably reduce the amount of venom injected. A study of the biting apparatus of elapine snakes by various authors (Acton and Knowles, 1914; Fairley, 1929*b*) has shown that the largest amounts of venom are injected when the lower jaw is fixed, and a snake of this family bites at a mechanical disadvantage when it strikes at a flat surface such as the calf of the leg.

The larger the dose of venom received, the shorter the death time (Lamb and Hunter, 1904*b*; Acton and Knowles, 1914). If the venom is injected intravenously or if a massive dose is injected subcutaneously, death may occur within half an hour of the bite (Acton and Knowles, 1914; Kellaway, 1942). Such occurrences are very rare, and death most commonly occurs within two to 48 hours of a venomous terrestrial snake bite. In cases reported in Australian literature, death has more commonly occurred towards the end of this period (eight deaths in less than three hours of the bite and 62 deaths after more than 12 hours out of a total of 81 fatal cases—Fairley, 1929*a*).

In seven of the cases reported above, we believe that death almost certainly would have occurred without treatment. There is no doubt in our minds that these seven patients received a dose of venom that would have proved to be a lethal one without treatment. In none of these cases, without treatment, would the estimated death time have been under eight hours, so probably none of the patients received a massive dose of venom. These cases are quite comparable in severity to a number of fatal cases of snake bite reported in the Australian literature (Ferguson, 1926; Fairley, 1929*a*; Flecker, 1940; Kellaway, 1942; Benn, 1951). They are not as severe as the cases reported by Flecker (1944). The other patients in this series received a sublethal dose of venom, or a lethal dose that was modified by antivenene given prior to admission to hospital (tiger snake antivenene in small or large doses over a period of up to 36 hours), and recovery undoubtedly would have occurred without hospital treatment.

The Clinical Effects of the Venom.

Although the venoms of poisonous Australian snakes, like those of other elapine snakes, contain several toxic

factors such as enzymes (Porges, 1953), the clinical picture following envenomation is dominated by the effects of the neurotoxins, and death is usually due to the action of these toxins (Fairley, 1929*a*; Kellaway, 1942). This particularly applies to the venoms of the death adder and the taipan (Kellaway, 1929*c*; Kellaway and Williams, 1929). Because of this, it is usually clinically impossible to determine the species of the elapine snake causing the bite from the symptomatology produced (Lamb and Hunter, 1904*b*; Lamb and Hunter, 1906; Fairley, 1929*a*; Kellaway, 1942), and the symptoms described above resulting from bites of Papuan elapine snakes are very similar to the symptoms which follow bites by the cobra and the krait, the main venomous elapine snakes of India (Lamb and Hunter, 1904*a*, 1904*b*).

There has been no published work on the venom of the Papuan black snake, but clinical observations suggest that death from bites of this snake may sometimes be due to anticoagulant or hæmolytic effects of the venom. If these observations are substantiated, and work on the venoms of other snakes of the same genus suggests that they will be (Kellaway and Williams, 1929; Kellaway, 1930), clinical signs may be of some assistance in indicating that bites from this snake have occurred.

The main effect of elapine neurotoxins falls on the motor component of the nervous system, where they appear to have a curare-like action at the neuro-muscular junction, but they may also have some direct action on the muscle itself (Kellaway, 1932; Kellaway, Cherry and Williams, 1932). This action at the neuro-muscular junction is complex (Porges, 1953) and is uninfluenced by neostigmine.

Clinically, the susceptibility of the extrinsic ocular and eyelid muscles and the resistance of the diaphragm to the action of the neurotoxins would seem to support this theory of a curare-like action. Some features of interest in their action are the possibly long latent period before the effect is apparent, the long duration of their effect and the occasional maximal severity of their peripheral effect in the bitten limb. The drowsiness probably represents a central action of the neurotoxins, and in two of the severest cases the patients have had amnesia for the events of the first few days of their illness, although they had apparently remained conscious and cooperative.

The ocular and the bulbar muscles are usually most severely affected by the action of the neurotoxins. The fact that death may occur from involvement of the bulbar muscles has not been sufficiently stressed in the Australian literature. Death must occur quite commonly from this cause. The jaw may drop back; the tongue is paralysed and cannot move, and respiratory obstruction occurs in a patient who cannot move his head significantly. The patient cannot swallow, and secretions accumulate in the pharynx and may be drawn into the lungs and trachea. Fairley (1929*a*) commented on the fact that the tracheæ and bronchi of sheep were packed with food debris after death due to tiger snake venom. Also, owing to the paralysed intercostals and the weak diaphragm, bronchial secretions cannot be coughed up. With an airway obstructed by secretions or by the tongue, breathing becomes laboured, and the weak intercostal muscles and the diaphragm may cease working at a much earlier stage than they would have done had the anoxia been corrected. In some of our cases the improvement in the patient's general condition and respiratory movements after tracheotomy and the insertion of a cuffed tracheotomy tube was dramatic. However, in the most severe cases, even if an adequate airway is maintained, and oral secretions are prevented from being aspirated into the trachea and lungs while endobronchial secretions are removed, death will occur from paralysis of the intercostal muscles and diaphragm if artificial respiration is not resorted to. When respiration is assisted, complete failure of the diaphragm occurs many hours after the first onset of its weakness.

Once the effects of anoxia are removed, the lack of involvement of the cardio-vascular system by the venoms is to be noted. Many workers, experimenting with various elapine venoms in animals, have commented on the fact that the heart continues to beat strongly for some time after the cessation of respiration and before death (Lamb and Hunter, 1904*a*; Acton and Knowles, 1921; Fairley, 1929*a*; Kellaway, 1929*a*, 1929*b*, 1929*c*; Kellaway and Williams, 1929). However, Kellaway (1942) thought that the cytolytic action of Australian snake venoms led to the liberation of histamine, and that symptoms of cardio-vascular failure after bites of Australian snakes were due to this. However, a study of some such case reports suggests that chronic anoxia and carbon-dioxide retention may have been the cause of these cardio-vascular symptoms.

In several cases in this series, there has been a mild anticoagulant effect from the venoms, and in one case a mild hæmolytic effect. Albuminuria was an inconstant feature of envenomation, as was also microscopic hæmaturia.

The Reversibility of the Muscular Paralysis.

If the patient survives, the muscle paralysis is usually completely reversible, even if an apparently lethal dose of venom has been received. This fact needs to be stressed. Acton and Knowles (1921) state that in the case of cobra bites in animals, paralytic effects, however severe, are rapidly recovered from if the animal survives. The same observation has been made by Kellaway (1929*b*) in the case of bites from the Australian copperhead snake, and by Fairley (1929*a*) in the case of bites from the Australian tiger snake.

Clinically, muscles that were completely paralysed usually remained so for two to five days, and then recovery commenced, the process of recovery continuing for another two to 10 days. The usual course is for the paralysis to clear completely within five to 14 days of the bite without any residual disability. We believe that this represents the normal course of this peripheral effect of the venoms, in spite of the antivenenes administered. In one of our most severely ill patients, who was very anoxic on admission to hospital, the paralysis persisted much longer, and some residual disability will remain (Case XIII). A long duration of the paralysis following a bite from an Australian tiger snake has been noted (Fairley, 1929*a*). Krait bites are also sometimes followed by chronic neurological lesions (Acton and Knowles, 1921).

A question of great practical importance is this: Does the paralysis ever reach an irreversible stage? And is recovery still possible after the injection of massive doses of venom? Kellaway (1932) thought that the prolonged direct action of the lecithinase in Australian snake venoms, which alters the permeability of muscle cells, was difficult to reverse, and concluded that there was no evidence that it could be reversed within the times covered by his experiments. In none of his experiments was the artificial ventilation of the paralysed animal continued for longer than 30 hours, and the technical difficulties of maintaining life for such a long period in a small animal were stressed by him. As muscles may remain completely paralysed clinically for two days or more before they show signs of recovery, one concludes that these experiments probably were not continued long enough to show definitely that the paralysis was irreversible.

There is need to prove or disprove this point experimentally for the various elapine venoms by the use of modern techniques of artificial respiration, cuffed tracheotomy tubes and intravenous feeding in suitable animals. Working with rabbits, a small and possibly unsuitable animal, Silberberg (1954) attempted to do this in some measure by injecting three times the minimum lethal dose of tiger-snake venom subcutaneously into the animals and then artificially ventilating them through a tracheotomy tube when respiratory failure developed. He found that the rabbits all died from cardio-vascular failure within 11 hours of the commencement of artificial respiration. He attributed this cardio-vascular effect to the circulatory depressant action of the venom. We have

not observed any such effect in humans bitten by Papuan elapine snakes, and the need for further experimental work still exists.

If severe anoxia is present, some permanent neurological damage may result from this and from the effects of the venoms. The post-mortem changes found in the nervous system after death from snake bite are usually attributed to anoxia (Kellaway, Cherry and Williams, 1932).

The Treatment of Snake Bite.

First-aid treatment contributed little, if anything, to the successful management of our cases, and measures such as incision of the bitten area and wrongly used tourniquets often added to the patient's discomfort without giving any benefit. First-aid measures of proven value in animals are the immediate application of an effective tourniquet plus the excision or amputation of the bitten part or venesection of the bitten limb (Fairley, 1929*a*; Kellaway and Morgan, 1931). To be effective, these first aid measures must be applied immediately after the bite is inflicted. The likelihood of this occurring in practice is very remote. Except for the application of a tourniquet, these measures are too radical and impracticable for field use. This is particularly so when it is realized that it is sometimes difficult to decide whether a person has been bitten by a venomous snake or not, and completely impossible to decide initially whether a bite from a venomous snake is going to be a serious one.

First-aid treatment should be limited to the immediate application of a properly applied and properly used tourniquet (released every 20 minutes and discarded after two hours) and rest for the patient. (probably impracticable suggestions for this country). It is perhaps of more importance to educate the people to recognize the first symptoms indicative of envenomation, and to realize the need for the urgent transport to hospital of patients with such early symptoms.

When the species of the snake responsible for the bite is unknown, correct treatment with specific antivenenes is difficult. Because of its slow rate of absorption and the large doses required, intramuscularly administered antivenene has no place in the treatment of snake-bite. Very large doses of several types of antivenenes were given intravenously, after admission to hospital, to most of the patients described here. In addition, several of the patients had received intramuscular or intravenous doses of tiger-snake antivenene before their admission.

In only one case was there any dramatic reversal of neurological signs within a few hours of administering the serum. This man (Case XI) had a slight ptosis only and was admitted 15 hours after being bitten. In two cases (Case V and Case XIV) in which hæmaturia or hæmoglobinuria was present, these symptoms ceased after the administration of Papuan black-snake antivenene. The neurological signs in these two patients were not obviously influenced by this antivenene. In all the other cases no definite benefits were seen to follow the administration of antivenenes. In three of these cases (Case I, Case II and Case XV) the paralysis continued to progress in spite of the antivenene. In the other cases the antivenene may have prevented further paralysis developing.

The antivenene was given late in the course of the illness in most cases, often eight or more hours after the bite. Acton and Knowles (1921) thought that antivenene should still be of value if it was given within two-thirds of the death time and Ahuja and Singh (1954) state that, in the case of cobra bites, it is never too late to administer antivenene. In a case reported in the Australian literature, antivenene appears to have been of value although given more than 12 hours after the bite (Knyvett and Molphy, 1959). In the cases reported by Tisdall and Sewell (1931) and by Lester (1957), the attribution of the improvement to the antivenene, given late in the illness, was probably mistaken. On the whole, the observed benefits of antivenene treatment have been disappointing.

Tracheotomy has been widely used recently in a large number of different conditions in which mechanical ventilatory obstruction or secretional ventilatory obstruction exists (Nelson, 1958). These two indications for its use may exist in elapine snake bite. The successful use of tracheotomy in severe poliomyelitis suggested that it should be of value in snake bite (Reid, 1956). Knyvett and Molphy (1959) reported a case of severe snake bite in which a tracheotomy was performed. In this case it was performed very late in the course of the illness which was complicated by uræmia, and the patient died. Our observations have confirmed its value in the treatment of severe snake bite. Fortunately, although we are working with incompletely trained staff, no complications of tracheotomy have occurred.

Since the experiments of the workers of the Indian Snake Poison Commission (Richards, 1886), the importance of artificial respiration in prolonging life in cases of serious elapine snake bite has been well established. By inserting cannulæ into the tracheæ of dogs and artificially ventilating them, they were able to prolong for up to 41 hours the lives of dogs who had received lethal doses of cobra venom. However, they concluded that, although of great value in supporting the vital functions for some time, the measure was of doubtful value as a means of saving life. With our modern techniques, artificial respiration can undoubtedly prevent a fatal outcome in some cases of serious snake bite, but may have to be continued for two days or more.

The nursing care and medical treatment of the completely paralysed patient can be more easily carried out when the patient's breathing is assisted by some form of respirator or breathing apparatus attached to the tracheotomy tube. The patient must also be easily accessible so that he can be postured frequently. Because of this we think a tank respirator is to be avoided. We have found a manually operated rebreathing bag with a soda-lime absorption unit to be more satisfactory than an intermittent positive pressure breathing apparatus, and when used for such an ill and helpless patient it is probably much safer. Some simple apparatus involving the use of an oxygen cylinder, a Waters cannister and a rebreathing bag (Lassen, 1956) could probably be employed in rural hospitals for the treatment of this condition. So long as regular supervision is given by trained staff, intelligent semi-trained nursing staff can cope in an emergency with this apparatus and this method of treatment.

Conclusion.

Owing to the difficulties of transport in Papua, the cases reported in this paper may represent a selected group, as the most severe cases may never reach hospital. In these very severe cases other toxic factors in the venom may become of more significance and present new difficulties in treatment. However, we believe that, with modern treatment, the elapine snake victim should not die if he lives long enough to reach a hospital, and if the neurotoxic effects of the venom are the main factors in his illness.

Reports of Cases.

Case XIII.—A male Papuan, aged about 25 years, was admitted to hospital at 10.30 a.m. on May 31, 1960, in a semi-comatose condition. He had been bitten by a snake at 4 p.m. on May 30. During the night before his admission to hospital he had been given 15,000 units of antivenene (? tiger snake) by intermittent intravenous injections. On his admission, he was found to be semi-comatose, restless, cyanosed and sweating profusely. His jaw was completely relaxed and had fallen back so that the tongue obstructed his airway. Direct laryngoscopy was performed immediately, and the vocal chords were seen to be moving. A cuffed endotracheal tube was passed. After an adequate airway had been established, the cyanosis and sweating disappeared and the restlessness decreased. On more detailed examination then, he was found to respond to painful stimuli, but

not to the spoken word. Complete ptosis of both eyelids was present. The eyes were centrally fixed. The pupils were unequal, because of an old iritis affecting the right eye. The left pupil was dilated and reacted to light. There was no movement of any of the facial muscles. The jaw muscles were completely paralysed, and the mouth remained open. No movement of the tongue or palate was detected. The patient could not swallow, and secretions accumulated in the pharynx. He could move his head slightly from side to side, but could not raise it from the bed. He could move all his limbs, but they appeared to be generally weak. There was very little movement of the intercostal muscles, but the diaphragmatic movements appeared normal and the patient had quite an effective cough. All the deep reflexes were present. The superficial abdominal reflexes were absent, and the plantar response was flexor in both feet. The pulse rate was 156 per minute and the systolic blood pressure was 130 mm. of mercury. There was a small wound above the left ankle, which was bleeding slightly. Examination of the urine showed a very heavy deposit of albumin. The hæmoglobin value was 10·4 grammes per 100 ml.; moderately high polymorphonuclear leucocytosis was present. No malarial parasites were seen in thick or thin blood smears. The prothrombin time was 67% of normal. The serum electrolytes were normal. Examination of the centrifuged deposit of the urine showed no abnormality. One and a half hours after his admission to hospital, a tracheotomy was performed under local anæsthesia. The oral endotracheal tube was removed and a cuffed tracheotomy tube inserted. An intravenous infusion of a 4% dextrose and fifth-normal saline solution was commonced and continued for six days; 30,000 units of taipan antivenene and 30,000 units of tiger-snake antivenene were given by injection into the tubing of the infusion apparatus. Intramuscular injections of 500,000 units of crystalline penicillin were given every six hours for one week. Two and a half hours after his admission to hospital, the pulse rate was 114 per minute and the blood pressure was 110/80 mm. of mercury. The patient later had a rigor and his temperature rose to 104° F.; he remained restless throughout that day, requiring sedation with paraldehyde. The temperature gradually returned to normal over the next four days. There was no significant change in the patient's condition until the morning of June 2, 1960, when very slight lateral movements of the eyes and the slightest flicker of movement in the eyelids were detected. On the evening of that day restricted but definite eye movements were present. The patient was still restless and uncooperative. On June 3 slightly more movement of the eyelids was present, the eyes opening one-eighth of an inch and moving one-eighth of an inch laterally to the right. On June 4 the eyes opened one-quarter of an inch, full lateral movement to the right being present. By June 5 no ptosis was detected.

The subsequent progress of the patient can be summarized briefly. The patient initially had paralysis of conjugate movement of the eyes to the left and was unable to move his head to the left of the midline, but these signs disappeared by June 11. On June 6 it was apparent that there was slightly more weakness of the left side of the face, and that the left arm was weaker than the right. This left-sided weakness gradually decreased, but still persists. He gradually became less restless, and by June 6 was able to obey some commands, but he still appeared confused. He gradually became more rational and cooperative, and by the end of June was mentally normal. On June 8 he sat up with assistance, and he walked with assistance on June 20. Tube feeding by means of a gastric tube was commenced on June 6 and continued for two months. The tracheotomy tube was removed on June 9. After very slight initial movements of the lips, jaw, tongue and palate, very little improvement occurred in these muscles for two weeks, and then extremely slow but definite improvement started. By July 7 the patient was able to smile slightly, open his mouth about half an inch and protrude the tongue one-quarter of an inch beyond the teeth. His palate moved slightly, and he could now say "ah". On August 9 he was able to swallow normally, and tube feeding was suspended. He now could open his mouth for about one inch, and the tongue protruded one inch beyond the teeth. He was phonating better. On August 23 there was still only a moderate degree of palatal movement present. The movement of the jaw was still slightly restricted. He could not articulate clearly, and his speech was generally unintelligible. Apart from the upper motor neuron weakness of the left side of the face, the movements of the face appeared normal. On June 5 slight periorbital œdema was noted, and a transient erythematous rash and swelling of the lips developed on June 6. On July 12 he developed fever and acute polyarthritis affecting the right knee and the right proximal interphalangeal joints. Some joint swelling and pain from this condition still persist.

This patient had complete paralysis of all the ocular, facial and bulbar muscles. This led to respiratory obstruction and severe anoxia. He had a moderate degree of intercostal paralysis, but no detectable involvement of the diaphragm. The anoxia was possibly complicated by a cerebral vascular accident, and there were several manifestations of serum sickness. The recovery from the illness has been very slow, and residual disability is certain.

Case XV.—A male Papuan, aged about 25 years, was admitted to hospital at 8.45 p.m. on August 5, 1960. He was unable to give any history, but an accompanying letter from the medical assistant in a rural hospital at Rigo indicated that the man had been bitten by a snake at approximately 12 noon that day. He had reported to the Rigo hospital at about 2.15 p.m., where he had been given 6000 units of tiger-snake antivenene and 9000 units of Papuan black-snake antivenene by intravenous injection before 3 p.m. He was then transferred by road to Port Moresby. After his recovery some additional history was obtained from the patient. While working in grass about a foot high, he was bitten above the left ankle by a black snake three feet long. The snake struck and let go immediately. After the bite he continued to work, but after some minutes he noticed a headache and then became dizzy. He became frightened, and ran about one hundred yards to his employer, who, about half an hour after the bite, applied a tourniquet a few inches below the knee and incised the wound. As the tourniquet was being applied the patient vomited some blood, and shortly after this he developed double vision. He felt sleepy and could not open his eyes. He remembers being brought to hospital but nothing after that. On examination the patient was restless and drowsy. He responded to some questions, but was uninterested. While being examined, he vomited a few millilitres of blood. Complete bilateral ptosis was present. The eyes were centrally fixed. The pupils were slightly dilated and reacted to light. The mouth could be opened to only half its normal extent, and the jaw was quite relaxed. The tongue could be protruded for about one inch beyond the teeth. There was a moderate degree of weakness of the palate. The patient appeared to be able to swallow, and his speech was slurred but intelligible. He was unable to lift his head from the pillow, but could move it from side to side. He could not sit up, but was able to roll his trunk from side to side and turn on to his side. There was generalized weakness of the limbs, affecting the proximal muscle groups more than the distal. The patient was unable to lift his legs from the bed. There was no movement of the intercostal muscles, and the diaphragm was weak. Pain sensation appeared normal. All the deep reflexes were present, and the plantar reflexes were flexor. There was a half-inch incised wound 4 in. above the anterior aspect of the left ankle, which was bleeding freely. There was slight œdema of the left foot. The temperature was 98° F. and the pulse rate was 90 per minute. The blood pressure was 140/90 mm. of mercury. A "grade 3" spleen was present. There was no albumin in the urine.

Papuan black-snake antivenene (21,000 units) was given intravenously soon after his admission to hospital. An intravenous infusion of a 4% dextrose and fifth-normal saline solution was commenced and continued for seven and a half days. Potassium chloride, in a dose of 8 grammes per day, was later added to this fluid. The patient's breathing became weaker and more obstructed, and an endotracheal tube was passed. A tracheotomy was then performed under local anæsthesia. A cuffed tracheotomy tube was inserted. A further 15,000 units of Papuan black-snake antivenene, 30,000 units of taipan antivenene, and 30,000 units of tiger-snake antivenene were given into the tubing of the infusion apparatus. Intramuscular injections of penicillin were commenced, but later the antibiotic was changed to "Chloromycetin". Four hours after the patient's admission to hospital the paralysis had progressed. The diaphragm was quite weak, but respiratory exchange was thought to be adequate. Bleeding continued from the leg wound and from the tracheotomy wound. One litre of blood was given during the night. By 9.30 a.m. on August 6, further extension of the paralysis had occurred. The diaphragm was very weak and breathing was assisted, the patient was cooperative, but he could not open his mouth at all. The tongue could not be protruded, but it could be moved slightly on the floor of the mouth. There was no movement of the palate. The patient could only wrinkle the forehead and could move the lips only slightly. His head movements were restricted to about half an inch on either side of the mid-line. He could not turn his trunk, but only twist his body slightly and ineffectually. The wrist and fingers could be flexed a little. The only movement in the lower limbs was in the

toes. The deep reflexes were absent. The œdema of the left foot was more marked. The urine contained a large amount of albumin. By 2 p.m. that day there were virtually no spontaneous respiratory movements, and at 5 p.m. there was no spontaneous breathing at all. However, by 12 o'clock midnight regular but very inadequate diaphragmatic movements had returned. The tracheotomy wound continued to ooze blood. On August 7 further extension of the paralysis in the limbs and face had occurred. The movements of the diaphragm remained weak, and were assisted all day. The patient could only raise the fingers and move the toes only slightly. There was only the slightest evidence of movement in the lips and forehead. However, in the eyes the first evidence of recovery was present. The patient could move his eyes about one-sixteenth to one-eighth of an inch laterally. Later this day the eyelids commenced to move, and they could be opened about one-eighth of an inch by evening, when slightly more movement of the tongue was evident also.

By August 8 further improvement was in evidence. The eyelids opened three-eighths of an inch, and the movements of the eyes were almost normal. The tongue could be protruded half an inch beyond the teeth. Breathing was still assisted, but the diaphragmatic movements had improved slightly. That night the eye movements were normal, and there was more movement at the wrists when gravity was eliminated. More movement of the lips and forehead was present. The patient tried to form words with his lips and to issue instructions. On August 9 the diaphragm movements were stronger, and artificial respiration was ceased that morning. There was only slight ptosis remaining in the right eye. The mouth could be opened about one inch, and the tongue could be protruded one inch beyond the teeth. Slight movement of the palate was present, and also weak movements of the lips, nose and forehead. The head was moved about two inches to either side of the mid-line, and more movements of the fingers, wrists and toes were present. That afternoon the patient was febrile and had a rapid respiratory rate. Although his chest was clinically clear, it was thought that he might be developing pneumonia and he was given regular intravenous doses of "Reverin" and his breathing was again assisted until the next morning. On August 10 he was still febrile. Radiological examination of the chest disclosed no abnormality. The tongue movements were normal. The movement of the jaw was still slightly restricted. There was more movement of the facial muscles, the wrists and the ankles. By August 11 the jaw movements were normal. However, the palate was still weak. He was still able to move his head from side to side and flex and extend his elbows and knees in the bed. The deep reflexes were still absent. By August 12 the facial movements were normal, but the palate was still weak. The arms could be raised above the head, and the hips, knees and ankles could be flexed and extended while on the bed. The deep reflexes were present. Later that day the patient could swallow fluid and sit up in bed with assistance. On August 13 the palate moved normally, and the patient was able to swallow. He could lift his extended legs off the bed. His temperature was normal. The tracheotomy tube was removed and the intravenous administration of fluids was ceased. On August 15 the patient was able to sit and stand without assistance, but weakness of the neck and proximal limb muscles still persisted. On August 24 he still had some weakness of the shoulder muscles and the muscles of the left leg, but he should recover without any residual disability.

SUMMARY.

1. The symptomatology, the signs, the clinical course and the treatment of 15 cases of Papuan elapine snake envenomation are described. Two cases are reported in full.

2. The main effect of the venoms of Papuan snakes is to produce motor paralysis of the ocular, bulbar, jaw, facial and spinal muscles. Significant sensory changes and primary circulatory abnormalities were not detected. A mild anticoagulant or a mild hæmolytic effect of the venoms was observed in a small number of cases.

3. The successful management of the most severe cases involved the use of tracheotomy, artificial respiration and antivenenes. Seven patients with severe paralysis, who probably would have died without attention, survived with this treatment.

4. The complete reversibility of the severe paralysis in potentially fatal cases of elapine snake envenomation and the importance of respiratory obstruction as a cause of death in this condition are stressed.

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Reviews.

Modern Gynæcology with Obstetrics for Nurses. By Winifred Hector and John Howkins, M.D., M.S. (London), F.R.C.S. (Eng.), F.R.C.O.G.; Second edition; 1960. London: William Heinemann Medical Books Ltd. 8¾" x 5½", pp. 236, with illustrations. Price: 17s. 6d. net (English).

MISS WINIFRED HECTOR is the principal tutor at St. Bartholomew's Hospital, London, and Mr. John Howkins is gynæcologist and obstetric surgeon. This small book is of special value to nurses in general training, because of the outstanding sections on nursing care, particularly in the gynæcological section. A sketchy review of the major gynæcological problems is given with an outline of some of the surgical procedures, but the nursing of the patients concerned is given in detail.

There are excellent sections describing the examination of the patient, the preparation of the instruments for ward examinations, the setting out of theatre instruments for operations, and detailed chapters on pre-operative treatment and post-operative care of patients in gynæcological wards. The nurse is warned of complications which may occur, and she is given a very clear plan of emergency treatment. There are one or two rather quaint details which seem to remain in nursing textbooks, but these do not in any way detract from the very up-to-date nature of this book.

The salient points are very well set out in the text with excellent printing and good paper, and the illustrations are line drawings which on first glance seem too simple. However, they emphasize the important points, and are on the whole very good.

As is characteristic of the English point of view, the gynæcological section takes up by far the greater part of the book. The section on obstetrics is very scanty, and leaves out many of the major problems, such as antepartum and post-partum hæmorrhage. The book is certainly not adequate as a textbook for nurses training in obstetric and gynæcological hospitals, but could be a very useful supplement to lectures for nurses undergoing their general training.

Lectures on Hæmatology. Edited by F. G. J. Hayhoe, M.A., M.D., M.R.C.P.; 1960. London: Cambridge University Press. 9¾" x 6", with illustrations. Price: 60s. net (English).

THESE lectures were delivered at a symposium on hæmatology held in December, 1959, at Cambridge, under the auspices of the University Post-Graduate Medical School. The audience comprised medical consultants, senior registrars and research workers having hæmatology as one of their major laboratory or clinical interests. The lecturers were well-known British hæmatologists, each speaking on a subject to which he has contributed considerable knowledge. The symposium could not have failed to be most stimulating to those fortunate enough to attend.

The editor and publishers deserve commendation for their efforts in producing this volume. Many reports of symposia are literal transcripts of the delivered papers, and often do not appear well in print. There is evidence here of considerable editing, so that each lecture is well written, remarkably free of typographical errors and liberally supplemented with tables and figures, and each has a good list of references. Each lecture reviews aspects of some expanding field of hæmatology. There are three lectures on anæmia, one on transfusion hazards, one on abnormal hæmoglobins, five on aspects of leukæmia and the myeloproliferative syndromes and three on hæmostasis.

The emphasis on leukæmia reflects the growing interest in this disease. Hayhoe stresses the value of cytochemistry in differentiating the three major varieties of acute leukæmia. Using the PAS reaction, Fuelgen reaction, Sudan black B staining and alkaline phosphatase and peroxidase reactions, he is able to classify most cases of blast-cell leukæmia. Correct classification may be important from the therapeutic viewpoint. Doll analyses various aspects of leukæmia, and concludes that there has been a real increase in incidence over the last thirty years. There is statistical evidence that leukæmia comprises several separate disease entities. Galton describes his experiences with drug treatment of chronic leukæmia. In a rather speculative paper, Kay discusses the present position of marrow transplantation in the treatment of leukæmia.

Quite a few provocative statements appear on these pages, and one feels that they were made deliberately to stimulate an experienced audience to participate in discussion. A transcript of the discussions that took place would have improved the volume, but would have added considerably to the editorial difficulties. For those interested in either the clinical or the laboratory aspects of hæmatology, the acquisition of this volume is the next best thing to attendance at the symposium.

Modern Trends in Diagnostic Radiology. (Third Series) edited by J. W. McLaren, M.A., M.R.C.P., F.F.R., D.M.R.E.; 1960. London: Butterworth & Co. (Publishers) Ltd. 9¾" x 6½", pp. 304, with many illustrations. Price: 102s. 6d.

THIS volume is the third of a series with this title, the last volume having appeared in 1953. It attempts, as the editor states in the introduction, to "record our knowledge of the advances made in the last seven years". The various chapters are written by British and Continental authors, all of international repute in radiology. Those chapters which have been translated into English have lost little in clarity or ease in reading.

Modern equipment, including various methods of image intensification, three-phase generators, electronic printers and xeroradiography, is discussed, and this section offers an interesting glimpse of the radiology department of the future. A chapter on protection gives a comprehensive summary of present knowledge of radiation hazards and the technical means whereby individual dosage and population gonad dose may be minimized, and the chapter on radiology in obstetrics is a reasoned and balanced view of a subject jolted badly out of perspective by the Stewart preliminary report. It is a pity that these two chapters, which could be read with interest and benefit by all who operate X-ray equipment and all who practise obstetrics, should be part of a volume otherwise written fairly exclusively for radiologists.

Chapters on functional studies of the heart, œsophagus and urinary tract, and on the use of radiology in research, offer a fascinating suggestion that the horizons of radiology may be receding more rapidly than ever. The present usefulness of the various methods of operative radiology, including selective renal arteriography, peripheral arteriography, pelvic venography and cavography and splenoportography, is discussed and the technique in each case is summarized. Other chapters include a review of newer methods of investigation and interpretation of findings in the lungs, the biliary tract and the kidneys, in hypertension, in the bones and soft tissues and in pædiatric radiology.

As the volume contains chapters from a number of different authors, it is inevitable that the standard varies slightly from chapter to chapter; but it is generally of a high order throughout. X-ray appearances are constantly related to pathology and disturbance of function, and there

is a gratifying absence of unsupported authoritative statement and ex-cathedra utterance. The publication is well produced. Of the radiographs, most are negative and some positive, and they are generally excellent; but in one or two instances the reproduction is insufficiently clear to illustrate the point. The last seven years' advances have been fairly summarized and presented with clarity. The book is full of interest for every radiologist.

Recent Advances in Pathology. Edited by C. V. Harrison, M.D.; Seventh edition; 1960. London: J. & A. Churchill Ltd. 8" × 5¼", pp. 472, with 140 illustrations. Price: 48s. net (English).

THIS book, perhaps better known to many pathologists as "Hadfield and Garrod", now appears in a welcome new edition by the Professor of Morbid Anatomy at University of London Postgraduate Medical School. The previous edition with Hadfield as editor appeared in 1953. This latest edition has a formidable group of super-specialists as contributors on particular subjects. Editorial interference is stated "to be kept to a minimum only trying to ensure that various chapters are understandable by ordinary non-specialist readers". One concurs with this intention; but some of the chapters are found to be difficult because of the inherent complexity of their subject matter.

Much of the contained information could be justly said not to be recent. Even so, it is topical, and serves as a timely reminder and revision of interesting subjects. For example, the section on the diseases of the kidney reminds us of the hazards of radiation nephritis, and the presence of morbid renal changes from adult pyelonephritis illustrates "a six times more common cause of granular contracted kidneys than glomerulo-nephritis". In this chapter there is a timely reiteration of the dangers of catheterization, with experimental proof that certain microbes can migrate on the external surface of indwelling catheters. The book contains many such reminders.

Other subjects, though not new, are arranged and presented in a different and usually clearer light. For instance the chapter on the diseases of bone emphasizes the dynamic aspects of the skeleton, which makes diseases such as osteoporosis and osteomalacia easier to understand, whilst the section on diseases of the arteries gives a rational summarized review of the mechanisms of the production of atherosclerosis and coronary thrombosis. Many of the contents are truly recent, and the leader learns of many new diseases and advances in knowledge.

The book is well set out, with a concise summary at the end of some sections and a bibliography at the end of each chapter. It will save much waste of time for those who seek information on the newer or remodelled diseases, and will put order into previously disordered thinking. It is well illustrated with black-and-white photographs, but has only one page of colour plates. In common with all books of this nature, it is too brief on some subjects and has some omissions. A chapter on histochemistry would be a welcome addition. In the main it is a great little book, well worthy of a place in the library of senior medical students, physicians and pathologists.

Practical Dietetics. Edited by William A. R. Thomson, M.D.; 1960. London: The Practitioner. 9¼" × 6", pp. 64. Price: 4s. 6d. (English).

DURING 1959 *The Practitioner* published a series of articles, entitled "Practical Dietetics". "The aim of the series was to provide the general practitioner with authoritative, yet practical, guides to the planning and supervision of those diets which may be necesary for his patients." These articles have been reprinted in booklet form. Each article has been written by a high-ranking dietitian, most being chief dietitians of various English hospitals. There are 12 articles—"Low Salt Diet", "Low Fat Diets", Low Residue Diets", "Diets in Renal Disease", "Diabetic Diets", "Slimming Diets", "Diets for Peptic Ulcer", "Diets in Toxæmia of Pregnancy", "Weaning Diets", "Diets in Gastroenteritis in Infants and Young Children", "Feeding Old People" and "Canteen Diets".

The articles are very practical, giving the kinds of food which can be taken or should be avoided, recipes and menus and brief accounts of the reasons for the selections. In some cases proprietary foodstuffs are recommended which are available in England but may not be in Australia; but this should not lead to much difficulty. As all this information is contained in 58 pages, it will be seen that there is not much padding or unnecessary verbiage. At 4s. 6d. (English) the book is very good value, and should be in the hands of every practitioner interested in the diets of patients. This is an excellent book.

A Short Textbook of Midwifery. By G. F. Gibberd, M.S., F.R.C.S., F.R.C.O.G.; Seventh edition; 1960. London: J. & A. Churchill Ltd. 8¼" × 5⅝", pp. 590, with 200 illustrations. Price: 36s. net (English).

THE seventh edition of this book follows the conservative pattern of previous editions. The subject matter covers the general field of obstetrics, with chapters on anatomy and physiology, normal pregnancy and labour and abnormalities and their management. There is a commendable chapter on puerperal infection, in which the morbid anatomy of this condition is discussed in some detail, in spite of the recognized fact that the natural course of the disease is seldom seen these days.

The basic teachings are sound, and will provide a reliable, but necessarily brief, guide to most problems. Few references to the work and opinions of other obstetricians appear in the text, but notable inclusions are Lovset's manœuvre in breech delivery, and the work of Macafee on placenta prævia and that of Theobald on the intravenous use of oxytocic principle in uterine inertia. The general impression is of very personal teaching based on experience and material from the author's own hospitals, and this extremely conservative approach will lead the serious student to seek further afield in his search for knowledge of recent advances in obstetrics.

Staining Procedures: Used by the Biological Stain Commission. Revised by H. J. Conn, Mary A. Darrow and Victor M. Emmel with the assistance of H. A. Davenport, Georgia Gwinner, B. P. Kaufmann, S. I. Kornhauser, J. B. Longley, R. D. Lillie, Charlotte Pratt and Frank H. Smith; 1960. Baltimore: The Williams & Wilkins Company. 9" × 5¼", pp. 304. Price: 55s.

THIS book is published under the auspices of the Biological Stain Commission, University of Rochester Medical Centre. The first edition was published in 1943, appearing in a loose-leaf form. This was periodically revised (1943-1955), and the edition eventually consisted of nine leaflets. However, this present publication is the first in which all the material has been gathered together as a "hard-cover" book, and is regarded as the second edition of the entire contents.

The primary object for the organization known as the Biological Stain Commission was to secure high-quality and reproducible results by users of dyes for biological purposes. Difficulties are not uncommon in reproducing results with different batches of dyes, even from the same manufacturer, and although the policy of the Commission is against rigid standardization of methods, as this is inclined to oppose progress, some phases of technique can be stabilized; this is most desirable.

One omission relates to the use of stains and staining formulæ for material to be examined for the presence of malignant cells, in particular Papanicolaou stain, a procedure which is becoming increasingly used.

The book comes in three parts: "Animal Histology", six chapters; "Plant Histology", three chapters; "Microbiology", two chapters. An extensive bibliography enables those interested to trace the origin of methods. Containing as it does such a wealth of information, this book should find ready acceptance.

Heart Attack: New Hope, New Knowledge, New Life for Those Who Have Suffered a Coronary Thrombosis and for Those Who Have Not but Wish to Avoid It. By Myron Prinzmetal, M.D., and William Winter, Patient, with a Preface by Walter C. Alvarez, M.D.; 1960. New York: Simon and Schuster. 8" × 5¼", pp. 256. Price: $3.50.

THIS book represents a rather new departure in medical writing. It is a joint undertaking by a well-known American cardiologist, who practises in Hollywood, and one of his patients who has had a coronary occlusion. In the main, it is directed to the actual sufferer from coronary artery disease and assumes the form of a long "pep talk". While the argument and description are directed particularly to the sedentary, overweight American male, there are many good lessons for all types of sufferers from cardiac disease who have been advised by their medical attendants to lose weight, stop smoking, avoid nervous tension and take more walking exercise. An attempt has been made to cover all possible emergencies, and one chapter, for instance, is entitled "Sex?—Of Course". Various potentially embarrassing situations are cited, such as the need to avoid offending a good cook without exceeding one's calorie allowance. The

elementary physiology and pathology to which the earlier chapters are devoted are of debatable wisdom for the British reader; but throughout the book a genuine attempt is made to help the patient to understand the reason for and the means of overcoming his restrictions without misery or confusion. A series of appendices containing recipes, food values, flavourings and other diet hints enhances the practical value of the handbook.

There is sufficient similarity between the incidence of coronary disease, favourite foods and dishes and the pattern of life in general between the American and Australian sufferer from coronary artery disease to make this little book of great value to the latter, as well as filling in many details and answering many questions which neither the patient nor the doctor thinks about in the consulting room.

Medical and Dental Aspects of Fluoridation. By W. A. Cannell, M.R.C.S., L.R.C.P., D.P.H., L.D.S., R.C.S.; 1960. London: H. K. Lewis & Co. Ltd. 8½" × 5½", pp. 136. Price: 15s. net (English).

THIS small text of 125 pages, which first appeared in 1960, is intended to "clarify some aspects of the problem of the prophylactic use of fluoride in dental caries". However, the text falls very short of the author's purpose. The eleven chapters are arranged in a sequence which does not allow a logical development of the subject, while five of the chapters (II, III, VIII, IX and X) contain either no reference to fluoride at all, or negligible or insignificant reference.

There are a number of inaccurate and misleading statements, in addition to the omission of most of the essential matter of the fluoride-dental caries hypothesis. For example, in Chapter I (page 2), a section entitled "The History of Fluoridation" is a brief statement of the development of the fluoride-caries hypothesis, a quite different subject. The research which showed that mottled enamel, or dental fluorosis, was in fact due to fluoride, is not mentioned, and the omission of reference to the classical studies of Smith, Lantz and Smith in the United States and of Velu in North Africa is remarkable.

The critical observation made by H. T. Dean, which is basic to the use of fluorides in the prevention of dental caries, is that there exists a level of fluoride in water which will significantly reduce dental caries, but which will not produce any undesirable "mottling" of the teeth. The author apparently does not regard this as sufficiently fundamental to state it explicity in the text, although it is the basis of his argument.

The chapter entitled "Material Medica and the Pharmacology of Fluoride" is devoted essentially to the physiological and pathological effects of fluorides. There has been a tremendous amount of laboratory, clinical and epidemiological research in this field which the author has not considered in this superficial review. Waldbott's statement concerning allergic response to fluoride, and Wilson's report of spinal involvement in Oxfordshire children, to take but two examples, have been considered most uncritically. Cannell fails to emphasize, as the British Ministry of Health did in its report (Note No. 9, July, 1955), that the statement that fluoride in drinking water (at 1·0 p.p.m.) is no hazard to health, is based on the "lifelong consumption of waters containing fluoride naturally, both in America and this country" (United Kingdom).

The formula (pages 78 and 80) for fluorapatite is wrong or a misprint, and carbonate apatite is mentioned several times as though its existence in biological systems was a confirmed fact. The author states that fluoride replaces calcium in the enamel apatite, a mechanism in support of which he quotes no evidence. Certainly the five known modes of fluoride uptake by hydroxyapatite do not include this particular reaction.

Cannell suggests that the relationship between fluoride ingestion and periodontal disease is a controversial matter; again no evidence is quoted to support this claim. However, Russell's very extensive investigations in the United States show that the periodontal tissues in adults or children are not harmed by fluoride.

The common misconception that fluoridation benefits only the children in the community is expressed on page 114. However, there is conclusive evidence that in adulthood protection against dental caries occurs, provided there is continuous exposure to fluoridated water.

The author concludes on page 117 that fluoridation should not be adopted as a matter of extreme urgency, even though dental caries is a serious health problem. However, this is in direct opposition to the conclusions of the New Zealand Commission of Inquiry on Fluoridation, which stated that widespread use of fluoridation should be made to achieve "an urgently needed improvement in the present serious state of dental health in New Zealand".

The uncritical acceptance of much of the material in this book could be extremely misleading, and it should be read only by those thoroughly familiar with fluoride metabolism and its dental implications. Even to such persons the book will be of very limited usefulness.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Biological Problems of Grafting: A Symposium", sponsored by the Commission Administrative du Patrimoine Universitaire de Liege and the Council for International Organizations of Medical Sciences, established under the joint auspices of U.N.E.S.C.O. and W.H.O. (March 18-21, 1959); 1959. Oxford: Blackwell Scientific Publications. 9¼" × 6", pp. 468, with illustrations. Price: 50s. net (English).

"The Chemistry of Thyroid Disease", by Rosalind Pitt-Rivers, M.Sc., Ph.D. (London), F.R.S., and Jamshed R. Tata, M.Sc. (Bangalore), Docteur ès Sciences (Paris); 1960. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications Ltd. 9" × 6", pp. 96, with figures. Price: 36s. (English).

"Culture, Society, and Health", edited by Vera Rubin; *Annals of the New York Academy of Sciences*, Volume 84, Art. 17, December 8, 1960. New York: New York Academy of Sciences. 9" × 6", pp. 783-1060, with illustrations. Price: Not stated.

"Physics and Electronics in Physical Medicine", by A. Nightingale, M.A., Ph.D., F.Inst.P.; 1959. London: G. Bell & Sons, Ltd. 8½" × 5½", pp. 306, with illustrations. Price: 30s. net (English).

"Miscellaneous Notes (Ninth Series)", by F. Parkes Weber, M.D., F.R.C.P., F.S.A.; 1960. London: H. K. Lewis & Co. Ltd. 7½" × 4¾", pp 8. Price: 2s. 6d. (English).

"Modern Treatment Year Book, 1961: A Yearbook of Diagnosis and Treatment for the General Practitioner", edited by Sir Cecil Wakeley, Bt., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S.; Twenty-seventh Edition; 1961. London: Baillière, Tindall and Cox, Ltd. 8½" × 5½", pp. 336, with illustrations. Price: 35s. (English).

"Neurovascular Compression Syndromes of the Shoulder Girdle", by Louis M. Rosati, M.D., and Jere W. Lord, M.D.; 1961. New York and London: Grune & Stratton. 9" × 6", pp. 176, with illustrations. Price: $7.25.

"World Directory of Venereal-Disease Treatment Centres at Ports", World Health Organization; Second Edition; 1961. Geneva: World Health Organization. 9¼" × 6", pp. 164. Price: 8s. 6d. (English).

"Deuterium Isotope Effects in Chemistry and Biology", edited by David Kritchevsky; *Annals of the New York Academy of Sciences*; Volume 84, Art. 16, November 25, 1960. New York: New York Academy of Sciences. 9" × 6", pp. 573-781, with illustrations. Price: $4.00.

"The Rickettsial Diseases", by P. F. Zdrodovskii and H. M. Golinevich; translated from the Russian by B. Haigh, M.A., M.B., B.Chir.; 1960. Oxford, London, New York and Paris: Pergamon Press. 8½" × 5½", pp. 644, with illustrations. Price: £5 5s. (English).

"A Laboratory Handbook of Blood Transfusion Techniques", by A. Derek Farr, F.I.M.L.T., A.I.S.T.; 1961. London: William Heinemann Medical Books Limited. 7½" × 4¾", pp. 148, with illustrations. Price: 17s. 6d. net (English).

"Second African Conference on Bilharziasis", World Health Organization Technical Report Series, No. 204; 1960. Geneva: World Health Organization, 9¼" × 6¼", pp. 38. Price: 1s. 9d. (English).

"Requirements for Biological Substances", World Health Organization Technical Report Series, No. 200; 1960. Geneva: World Health Organization. 9¼" × 6¼", pp. 32. Price: 1s. 9d. (English).

"Medical Supervision in Radiation Work", World Health Organization Technical Report Series, No. 196; 1960. Geneva: World Health Organization. 9¼" × 6¼", pp. 32. Price: 1s. 9d. (English).

The Medical Journal of Australia

SATURDAY, APRIL 1, 1961.

AWARD OF THE GOLD MEDAL TO DR. H. C. COLVILLE AND DR. W. F. SIMMONS.

THE Federal Council of the British Medical Association in Australia at its meeting in Sydney on March 17, 1961, awarded the Gold Medal of the Association in Australia to Dr. H. C. Colville and Dr. W. F. Simmons "for long and distinguished service to the British Medical Association and to members of the medical profession throughout Australia".

This is fitting indeed. Dr. Colville is President of the Federal Council, having filled that role with distinction since the death of his predecessor in office, Sir Archibald Collins. In the many roles that the President is called upon to fill — chairman, adviser, arbitrator, negotiator, spokesman, host, *raconteur*—he has given freely of his time and energy and of his store of wisdom, experience and humanity. His record of service is long. For 32 years a member of the Council of the Victorian Branch of the B.M.A., he has been its chairman for 22 years, and was President of the Branch in 1940. He has been a trustee of the Medical Society of Victoria for 15 years. Elected to the Federal Council as a Victorian Branch representative 21 years ago, he was elected Vice-President of the Council in 1951 and President in 1955.

Dr. W. F. Simmons has been a representative of the N.S.W. Branch on the Federal Council for 23 years and has been Treasurer of the Council since 1945. He has been the representative of the Federal Council on the National Health and Medical Research Council since 1943, and in 1957 became the Chairman of the Medical Research Advisory Committee of that Council. He has also been a trustee of the Medical Officers' Relief Fund (Federal) and of the Federal Medical War Relief Fund since 1943. In New South Wales Dr. Simmons has served the profession for over 40 years. He was honorary secretary of the Illawarra Suburbs Medical Association from 1920 to 1929 and has been a member of the Council of the N.S.W. Branch of the B.M.A. for 27 years, holding the office of President of the Branch in 1942 and 1943 and being Treasurer since 1957. He has been a Director of the Australasian Medical Publishing Company Limited since 1947 and a Director of the British Medical Agency of N.S.W. Limited since 1942.

Both Dr. Colville and Dr. Simmons are held in high regard by their colleagues, not only for their willing and capable service to the medical profession, but also for their personal and professional qualities. The decision of the Federal Council to bestow on them the Gold Medal of the Association in Australia will have warm and general approval. This will bring the total number of Gold Medals so far awarded to 10. The previous recipients have been W. T. Hayward, R. H. Todd, W. H. Crago, H. C. Mollison, A. Graham Butler, Sir Henry S. Newland, John G. Hunter and Mervyn Archdall.

THE AUSTRALIAN SCIENTIST.

AT the thirty-third meeting of the Australian and New Zealand Association for the Advancement of Science (A.N.Z.A.A.S.) held at Adelaide in August, 1958, Professor Sir Mark Oliphant delivered a Presidential Address[1] entitled "Science and the Survival of Civilization" which deserves to be read and pondered by all those who are concerned about the progress of our civilization. In the course of this address he berated his fellow scientists for their failure to keep in touch with their fellow men, and laid great stress on the urgent need for scientists to make their work understood by the general public. In a civilization whose economic life is founded, nurtured and nourished on the fruits of scientific discovery, it is of the greatest importance that those who conduct the affairs of the nation should possess an understanding of scientific thought and an awareness of what the scientists are about. In his opening remarks to the A.N.Z.A.A.S. conference Oliphant said: "unless science becomes of humanity and is felt as well as appreciated with intellect, it may well provide the means for mankind to destroy his civilization", and he concluded his address with the statement:

> I am not diffident about those suggestions. I put them forward with all the emphasis I can command, . . . because I am convinced that something along these lines must be done, and quickly, if this country is to be governed by men alert to the problems of the time, and elected by informed and intelligent citizens.

The suggestions referred to concern ways and means of getting across to the ordinary man something of the significance of recent advances in science and of making him aware of the basic nature and meaning of scientific activity, and we have little doubt that the appearance last month of a new magazine, *The Australian Scientist*, is the direct result of processes set in motion during Professor Oliphant's presidency of A.N.Z.A.A.S. in 1958-59. *The Australian Scientist*[2] is described as a monthly journal on research, development and application, and is published under the editorship of Dr. E. P. George, Director of the Physics Department of St. Vincent's Hospital, Sydney, assisted by an editorial board which includes such well-known names as Professor H. Messel, Sir Mark Oliphant and Sir Samuel Wadham. On the medical side we note the names of Professor A. A. Abbie of Adelaide and Professor R. H. Thorp of Sydney. In a letter accompanying our copy of the first number it is stated that this new journal aims to stimulate the interest of the responsible members of our community, that it will not specialize in any particular field but will cover science in its broadest sense, and that it is written on Leaving Certificate level. After reflecting on the last proposition, we concluded that an intelligent student of Leaving Certificate standard

[1] *Aust. J. Science*, 1958, 21, Number 4A (November).

[2] *The Australian Scientist*, Sydney: Tasman Productions. 8½" × 11¼". Subscriptions, £4 4s. per year, £7 7s. for two years, £10 10s. for three years.

who had taken science subjects should be able to read and understand most of the content of the articles in this first number, but they are not light reading. This magazine is for those whose interest is sufficient to induce them to further their scientific education by some serious reading. Those who take up the first number of *The Australian Scientist* in this spirit will find themselves well rewarded, for the ten original articles which it contains cover a wide range of topics, and each is written by a recognized authority in his field. J. M. Bennett contributes an article on "Trends in Automatic Computation"—a subject which is a closed book to most of us, but which is of the greatest practical importance in current moves towards automation. Under the title, "The Inconstant Constant", A. S. Fraser gives us an insight into one of the problems of modern genetics. In a paper of more general interest, "Life on the Seashores of Australia", Isobel Bennett, a leading authority on the zoology of the Australian coastline, presents a most readable introduction to the study of the life of this fascinating zone. Modern physics is well represented by an article on cosmic radiation by C. B. A. McCusker, one on plasma physics by A. W. Norton and one on the cyclotron by W. I. B. Smith. Medicine is represented by two articles, "The Function of the Mast Cells", by G. T. Archer and "Drugs and the Heart", by Professor R. H. Thorp. Under the title "New Deal in Science Education", Profesor Messel tells the success-story of the Summer Schools for Science Teachers organized by the University of Sydney School of Physics; this is a stimulating account of another move towards the wider dissemination of a real understanding of current scientific activities. There is also a foreword by the Honourable D. A. Cameron, Minister for Health, a pen portrait of Sir Macfarlane Burnet as Australia's first Nobel Prize winner, and, at the end, the first of a series of "Mathematical Recreations" by S. T. Butler for those who can enjoy mathematics as a form of relaxation. The magazine as a whole is beautifully produced and is a worthy example of the printer's art. It is printed on art paper, in a comfortable large magazine size, and the illustrations are excellent.

All the articles in this first issue are of Australian origin, and the intention is that in future 75% of all material used will be from Australian authors. To maintain this level of excellence will make considerable demands on Australian scientists, but the members of the Editorial Board are evidently confident that this can be done. We sincerely hope that their efforts will be well received, for their aims are certainly worthy of the fullest support. To try to bring science to the citizen is always an ambitious project. Recently, a worthy attempt to do this at a popular level has been abandoned, presumably for lack of public support—we refer to "Science News" and "New Biology", which were brought out at about quarterly intervals for a number of years by Penguin Books, and which contained many excellent articles on varied scientific topics, but which ceased publication last year. The popularization of knowledge is no new task, and those who undertake it perform an important service. James Jeans' "The Universe Around Us" and Lancelot Hogben's "Mathematics for the Million" were excellent examples in their time, but as the complexity of our society grows and the steady stream of new scientific discoveries forces fresh problems upon us, the need for the widest possible understanding of the scientific world by the community at large becomes ever more urgent. The production of *The Australian Scientist* is only one step in an attempt to meet this need, but it is a step in the right direction, and we wish it well.

Current Comment.

THE ANONYMOUS MYCOBACTERIA.

An unusual monograph[1] of 160 pages, "with contributions by 22 authorities whose skills and experience are outstanding", as the dust-cover modestly states, is built up from the verbatim reports of a conference held at the University of Texas. The subject is covered fully, in somewhat racy style—which makes for easier reading—by a series of lecturettes, case reports and panel discussions. Undoubtedly two of the guest speakers have attained to world eminence in this fascinating but as yet undeveloped subject. They are Dr. Ernest Runyon from Utah, who is responsible for the presently accepted four-group but still tentative classification of what are really "unclassified" mycobacteria, and Dr. Emanuel Wolinsky, who has made a prolonged study of these organisms in Ohio. The other participants are all from Texas, where there appears to be a remarkably high incidence (about 8%, as opposed to 2% in Ohio) of recovery of these mycobacteria in cultures from sputum or gastric washings.

Professor Chapman, Assistant Dean for Post-Graduate Education in the University of Texas, who has edited this slender but attractively set-out volume, in a foreword asserts that the time is ripe to pool the available information and assess its value. He points out the curious paradox that, though the groups of organisms are known, it is still not clear which are pathogenic; in short, the ætiology has been recognized in advance of the resultant diseases.

There are, of course, many different kinds of mycobacteria beside human tubercle bacilli; but, as Runyon points out, it is only recently that we have had convincing evidence of the error of assuming that the presence of acid-fast bacilli in sputum indicates tuberculosis. These newer groups, the photochromagens and so forth, are undoubtedly pathogenic in some cases, but there is no evidence that they represent mutations. Hence the term "atypical" in fairly general use is deplored, as they are not atypical tubercle bacilli.

The basic bacteriology is well presented in terms of isolation, identification and transmission, which, it is surprising to note, appears to be from either animal or soil to human host; no evidence of family infection has been discovered. Skin testing reveals a complicated picture, cross-reactions occurring in 80% of tests to human tuberculin and to photochromogen antigens, but oddly there are no cross-reactions between the anonymous groups. Incidentally, this has posed a diagnostic problem in many countries, and notably so in Queensland, where a fantastic tuberculin conversion rate round 80% in the north has so far baffled the public health authorities in that State.

An attempt to tabulate the special radiological features in the acute and chronic forms of the diseases that these mycobacteria presumably cause is somewhat tenuous, nor can the clinical pictures offered be regarded as characteristic at this early stage. But as pioneering efforts they are valuable.

[1] "The Anonymous Mycobacteria in Human Disease", edited by John S. Chapman, M.D.; The Sixth Annual Ivan H. Mattson I Memorial Conference of the University of Texas South-Western Medical School and Dallas Tuberculosis Association; 1960. Springfield: Charles C. Thomas; Oxford: Blackwell Scientific Publications Ltd. 9" × 6", pp. 194, with illustrations. Price: 60s. (English).

Therapy, it is made clear, is far from satisfactory, owing to resistance arising early. Essentially it is that of tuberculosis, and surgery may be needed to "cure" cavities, though it is agreed that if the disease is not infectious and lesions appear static, as tends to happen, they may be better left alone, as complication rates are rather high.

Though this problem of the unclassified mycobacteria is a real and possibly important one, especially to public-health officers and bacteriologists, it has yet to be placed in proper perspective. Already in two States of Australia, Queensland and Western Australia, it has been recognized, and is causing headaches to the tuberculosis divisions. Presumably it will arise in the other States, when thorough search is made. Meanwhile this book is commended to all who are interested in the subject, as it gives a very valuable conspectus of the present position. If we were to offer a note of criticism, it would be that a complete bibliography of all the papers—and there are many—published so far on the subject could have been included for reference. Perhaps the editor was in a hurry to publish while the news was still "hot". So let us not be carping; he has done a good job.

HEALTH INSURANCE.

No thoughtful doctor can fail to realize that the success or failure of voluntary health insurance in Australia will determine to a major degree his future form of practice. While, in the last resort, the fate of voluntary insurance will depend upon whether a private enterprise or collectivist philosophy predominates in the community, the satisfaction of a major sector of the population with this form of protection against the financial hazards of physical disability will be an important defence against revolutionary change.

These reflections are prompted by the reading of an important new book on health insurance by Edwin J. Faulkner.[1] Though this clearly written and comprehensive work is based entirely on American experience, much of it is directly applicable to the Australian scene, while the carefully considered principles of insurance have a universal application.

It is probably fortunate that in both countries the development of health insurance has had a large social service content, which has caused it to depart substantially from fundamental insurance principles. Thus both the Blue Cross and Blue Shield Associations in America, as also the Australian registered organizations, have always insisted on community rating, under which a flat-rate premium is charged to all members, on the theory that the good risks must subsidize the poor risks, lest the latter, if compelled to pay a premium adequate to their hazard, might find voluntary insurance priced beyond their reach. As a matter of pure insurance this is inequitable to the good risk, and in America life assurance companies have invaded this field, and by careful selection of risks and appropriate premiums have enticed many of this favourable group away from the Blue Cross and Blue Shield, leaving them with a more hazardous membership and a most unfavourable claims experience. They have only one real defence—to abandon community rating and fight the life companies on their own grounds with variable group ratings based on claims experience. This has already commenced, and is likely to spread widely.

In Australia, where the provision of benefits is a combined fund-Commonwealth venture, the system of community rating is being preserved by the Commonwealth additional benefit keeping premiums low in relation to the combined benefit, and by the device of special accounts, which skims off the more hazardous risks from the general insurance pool. The social service aspect of Australian health insurance can be well illustrated by one example. A primary basis of all insurance is that the loss (in this sense financial, due to injury or disease) must be accidental, in the sense that it is fortuitous and unpredictable, and yet may possibly be sustained by any contributor to the pool. How can normal pregnancy fit into such a postulate? Something like 10% of all benefit is being paid for a "loss" which, for perhaps the majority of contributors, is a biological impossibility. It is little wonder that in America "most insurers agree that the expenses of normal childbirth cannot be efficiently covered by insurance and could be more economically paid by the insurer himself through the family budget".

Every aspect of the mechanics and statistics of insurance is fully covered in this work. It ends with a chapter on "Trends and Conclusions", in which the immense difficulties facing voluntary health insurance in America are discussed. In this country, almost all of them are solved by operation of the happy partnership of Government with private enterprise insurance.

This book can be strongly recommended as required reading for health administrators, and for those doctors concerned with the management of health insurance funds in Australia.

[1] "Health Insurance", by Edwin J. Faulkner; 1960. New York, Toronto and London: McGraw-Hill Book Company, Inc. 8" × 5¼", pp. 648. Price: $8.75.

THE INFLUENZA EPIDEMIC IN ENGLAND AND WALES, 1957-1958.

A USEFUL REPORT of 72 pages[1] records Britain's experience of the 1957 pandemic of influenza. The epidemiological features are well presented, while the clinical studies and laboratory findings are most instructive. There is a brief description of limited trials carried out with various vaccines, and the report concludes with practical observations on future strategy against epidemic influenza.

The 1957 pandemic originated in China, reached Singapore early in May and India a fortnight later, and arrived in England by the middle of June. The virus was first introduced into England by air, and soon afterwards by sea travellers. The subsequent evolution of the epidemic conformed with a familiar pattern. Several initial "seedings" were followed by localized outbreaks, which in turn gave rise to others, and these eventually merged into a general epidemic, which is estimated to have involved over 7,000,000 people, of whom about 30,000 are believed to have died. (In contrast, the 1918 epidemic is thought to have killed about seven times that number.)

Infectivity was high, and clinical attack rates approached 50% in some groups. The brunt was borne by school-children and young adults. In compact communities the main epidemic span was three to five weeks. The disease itself was mild, and the commoner antipyretics with linctuses sufficed for home treatment, antibiotics being used selectively. The results of the vaccine trials are not easy to interpret, but suggest that substantial protection can be expected from timely vaccination with strain-specific preparations.

The observations concerning future strategy, which are contained in the concluding section of the report, are of special interest. On the assumption that the spread of influenza virus cannot be prevented and that vaccination affords the only safeguard against the disease, it is suggested that plans for future control should focus on delaying progress of the virus and urgent preparation of a specific vaccine followed by speedy immunization beginning with priority groups.

Influenza is perhaps the last remaining pestilence which has so far defied conquest. Three pandemics have ravaged mankind within living memory (1889-1890, 1918-1919, and 1957-1958). Until effective means of prevention are universally available, this disease must command the earnest attention of all who are professionally concerned with it. The British report has much to convey. It should be widely read.

[1] "The Influenza Epidemic in England and Wales 1957-1958", Ministry of Health Reports on Public Health and Medical Subjects, No. 100; 1960. London: Her Majesty's Stationery Office. 9½" × 6", pp. 76. Price: 4s. 6d. (English).

Abstracts from Medical Literature.

RADIOLOGY.

Fracture of the Orbital Floor.

J. R. Lewin *et alii* (*Amer. J. Roentgenol.*, April, 1960) state that the incidence of depressed fractures of the orbital floor is probably greater than many radiologists realize. These important injuries are frequently overlooked in cases of facial trauma because of the observer's lack of awareness that such fractures may be present although the orbital rim itself is intact. This type of fracture is caused by a sudden increase in intra-orbital pressure occurring when an object hits the front of the orbit, and, with or without rupturing the eyeball, forces the orbital contents backwards. In this type of injury, the thin medial orbital wall may also be fractured, or occasionally the medial wall alone is fractured. In the present authors' series, blows from clenched fists lead by far in being the chief offender in the production of this injury. Prompt diagnosis is important, since early surgical reduction of the fracture will reduce the likelihood of permanent diplopia, and will prevent a disfiguring degree of enophthalmos. The orbital floor is visualized as a slender curvilinear osseous density depressed into the attic of the antrum to varying degrees. Just superior to the displaced osseous fragment or fragments may be visualized a soft tissue density which represents downward prolapse of the œdematous inferior orbital contents.

Radiological Methods in the Management of Congenital Dislocation of the Hip.

A. F. Barrett and G. I. Verney (*Brit. J. Radiol.*, November, 1960) discuss the radiographic methods in use at the present time in the management of congenital dislocation of the hip. It has long been appreciated that a single radiograph in the antero-posterior projection is inadequate for excluding dislocation of a joint, and so reliance has to be placed on one of the other methods until stability of the joint is assured. The lateral view is virtually impossible to obtain when the patient is in a plaster cast. Stereoscopic antero-posterior radiographs are not usually procured by orthopædic surgeons, perhaps because of the difficulty in viewing and in fusing the stereoscopic images. In theory antero-posterior radiographs with tube shift is a good method for antero-posterior localization but in practice identification of the femoral head through plaster is frequently difficult. Moreover, both views are oblique, and if there is infero-superior as well as antero-posterior displacement, accurate localization is further complicated. Also, the method depends upon comparing the abnormal side with the normal and is only applicable in unilateral dislocation. In the authors' experience multisection tomography undoubtedly gives the best demonstration when the patient is in a plaster cast, and it is not difficult to perform. Only one exposure is required, and movement by the patient between exposures, which causes so much difficulty in stereoscopic and the tube shift method, is eliminated. Even in dysplastic hips with poorly ossified heads, and despite thick plaster casts, accurate assessment of the femoro-acetabular relationship can be made. A subluxation of as little as 1·0 cm. in the antero-posterior direction can be appreciated by the difference of definition of the femoral head in the three cuts.

Epidermolysis Bullosa.

M. Hadley and A. F. MacDonald (*Brit. J. Radiol.*, October, 1960) describe the radiological changes occurring in a case of epidermolysis bullosa. The authors state that the diagnosis of the condition is primarily a clinical one, but that the radiologist should be aware of the changes associated with the skin lesions and the various facets with which it may present to him. In the present case there was partial obstruction to the flow of barium down the œsophagus in its lower third due to a smooth tapering constriction about 3 cm. long with increased thickness of the œsophageal wall. Further radiological examination showed small frontal sinuses, a high arched palate, hypoplasia of the mandible and prognathism. In the hands there were hypoplastic metacarpals and phalanges, decrease in the width of the palms, complete absence of nails and very small spots of calcification in the tips of two fingers.

The Delayed Nephrogram in Ureteric Obstruction.

L. M. Dugdale (*J. Coll. Radiol. Aust.*, December, 1960) defines the delayed nephrogram as a phenomenon in which contrast medium which has been injected parenterally for pyelographic purposes accumulates in the parenchyma of the kidney, and later is slowly excreted into the calyces and pelvis. The appearance of the nephrogram suggests that the contrast medium is present in the tubules and possibly in the tubular cells. A kidney which shows poor function on intravenous pyelography may, in fact, be a relatively normal kidney in which the function has been temporarily impaired by obstruction of the major urinary ducts. It is the author's routine to continue an intravenous pyelogram examination up to 24 hours, in cases in which there is no excretion or only minimal excretion of radio-opaque contrast media by the kidneys visible in three-minute and 18-minute films. Occasionally a second injection is given about one hour after the first to reinforce the contrast obtained. Once the nephrogram has appeared, further films will show the slow passage of the contrast medium into the calyces, pelvis and ureter to give a pyelogram, although a wait of several hours may be required before sufficient is present to demonstrate the site of obstruction. Prone films, as well as those taken after the patient has been erect for a few minutes, are often of value, as the relatively dense contrast medium tends to sink through the urine into the dependent portions of the pelvis and ureter. The rate of disappearance of the contrast medium is very variable; there was no radiographic evidence of any contrast medium remaining in the pelvis in one case after 12 hours, while in others there was still quite good contrast at 24 hours. The author concludes that in any case in which a kidney does not appear to excrete the contrast medium as rapidly as normal, a prolonged examination is warranted because, if a nephrogram appears, this shows that the kidney function is but suppressed by a ureteric block. After a sufficient wait, the contrast medium will enter the pelvis and ureter, and in most cases reveal the site of the lesion, though not always its nature.

Diffuse Œsophageal Spasm and Muscle Hypertrophy.

A. S. Johnstone (*Brit. J. Radiol.*, December, 1960) discusses diffuse spasm and diffuse muscle hypertrophy of the lower part of the œsophagus. He states that radiological diagnosis in these conditions is based chiefly in the departure from the accepted sequence of movement set up by swallowing. In the normal person fluid barium passes rapidly down the œsophagus in a uniform column well ahead of the final wave of peristalsis which follows at the rate of about 4 cm. a second, clearing away the residues of the bolus. The œsophagus expands in a smooth uniform manner, and on returning to its resting state the fine longitudinal folds are outlined by traces of barium. Variations may be observed in the rhythm of contraction in the lower half, for at times it appears to contract in a single movement rather than in slow progression. Contractions are defined as abnormal if they depart from the above description; for example, the column may be held up by a single spasm, or be broken up by multiple spasms—the smooth contour of the distended œsophagus may become crenated by shallow ring constrictions. At times the barium column is broken up into a chain of spheres, some perhaps projecting eccentrically and, more rarely, one may find large circular dilatations. The shapes may wax and wane rhythmically until all the medium is cleared away. There are no set rules governing radiographic technique for the demonstrations of abnormal contractions. The consistency of barium does not usually influence their production, though at times a solid form of barium capsule will bring out an abnormal spasm not revealed by fluid medium. The position of the patient in most cases does not affect the result of the examination, but when he is lying supine the slower rate of movement makes for earlier recognition of slight abnormalities. However, it must be appreciated that in this position the œsophagus becomes a little redundant, especially if a hiatus hernia is present, and kinking may resemble abnormal contractions. The most striking examples of incoordination occur during the phase of restoration, and the abnormalities appear in the segment which lies between the aortic arch and diaphragm. The clinical assessment of abnormal movements presents a difficult problem but two guiding factors have been proved of value. These are (*a*) a history of dysphagia or attacks of retro-sternal pain, and (*b*) the radiographic demonstration of wall thickening, which undoubtedly indicates an abnormal condition. A thickened wall

may be recognized by a line running parallel to the column of barium except where the lumen may suddenly narrow. Here the line will continue in its original direction, indicating further thickening at the site of constriction. The clarity of the line varies with technical and physical factors and is best seen when the heart or spine lies in the background. A wall shadow is only rarely seen in the radiograph of the normal œsophagus, but it always occurs in cardiospasm and is a valuable sign, particularly in the early stages. It should not be assumed that a well-defined wall shadow means muscle hypertrophy; a similar appearance may be found with malignant infiltration and in peptic œsophagitis. The evidence nevertheless indicates that abnormal contractions and muscle hypertrophy are closely related.

Periureteric Fibrosis.

H. L. TWIGG (*Amer. J. Roentgenol.*, November, 1960) describes periureteric fibrosis as a clinical entity characterized by a retroperitoneal inflammatory mass having a definite histological picture and producing a progressive obstruction of the upper part of the urinary tract. There are several consistent findings on the pyelograms that should be mentioned. First, there is hydronephrosis with a hydroureter in the upper third. This dilated portion tapers gradually to a smooth cone-like narrowing in the mid-ureter at the point of the segmental constriction. This portion of the ureter is frequently pulled towards the mid-line and may even lie medial to the lateral border of the vertebral bodies. As the process rarely advances below the brim of the pelvis, the distal portion of the ureter is normal. Good ureterograms are essential, for inadequate filling of a hydroureter can simulate the disease. At operation, the process is seen as a dense fibrous plaque which may involve only a small segment of the ureter or may extend all the way from the kidneys to the sacrum. The area between the bifurcation of the aorta and the brim of the pelvis is the site most commonly involved. The ureter is surrounded and compressed by the mass, and the fibrous tissue may actually invade the wall. The mass rarely extends significantly lateral to the ureters. The ætiology of periureteric fibrosis is unknown. Many suspect it to be idiopathic. Others consider it a vasculitis or collagen disease. The frequent association of pyelonephritis with periureteric fibrosis is striking and points to an infectious etiology. However, this may well be the result of the obstruction rather than a cause of it. A consideration of the anatomic area involved shows us that the abdominal aorta, vena cava and ureters lie within a fascial compartment. This is formed by the renal fascia which is continuous laterally with the sub-peritoneal fascia. Medially, it divides into an anterior and posterior lamina, which unite with the same layers on the opposite side. Within this same space are the paraaortic lymph nodes which receive drainage from the genito-urinary organs as well as from the gastro-intestinal tract through the mesenteric system. Thus it is possible that infection or tumour from many different sources may initiate an excessive reaction in this area producing a fibroblastic proliferation. This may be viewed as analogous to the hyperplasia of scar tissue in the formation of a keloid. Hence, periureteric fibrosis may be looked upon as an exaggerated local response to many different stimuli, whether infection, tumour or trauma.

RADIOTHERAPY.

Myelomatosis.

J. F. BROMLEY (*Clin. Radiol.*, January, 1961) reviews a series of 87 cases of myelomatosis (plasma-cell myeloma) registered with the Department of Medical Statistics of the United Birmingham Hospitals during the years 1950 to 1956. One patient is alive at three years without any treatment. Five others are alive at four, five, six, eight and nine years respectively treatment by X rays. The maximum incidence is in the age group 60 to 70 years (31 cases) with the age group 50 to 60 years next (21 cases). Backache was the first symptom in 40% of cases, with "other bone pains" in 12·5% of cases. Chronicity is often a feature of the disease, and useful life can frequently be prolonged for many months and even years if the patients can be kept ambulant and reasonably free from pain. Twenty-two patients received no treatment at all. Their median survival time was two months. Seventeen were treated by urethane with a median survival time of eight months. Thirty-one were treated by X rays with a median survival time of 26 months. The respective survival rates at two years are 4·5% for no treatment, 5·9% for urethane, and 58% for X-ray therapy. The corresponding figures at three years are 4·5% for no treatment, nil for urethane, and 38·7% for X-ray therapy. The author states that the disease is more common than was formerly supposed and should be suspected in the presence of a history of severe pains in bone accompanied by profound anæmia, which should call for investigation of the serum proteins and examination of sternal marrow for the typical tumour cells.

The Chemotherapy of Multiple Myelomatosis.

R. C. S. POINTON (*Clin. Radiol.*, January, 1961) reviews the use of various chemotherapeutic agents in the treatment of multiple myelomatosis and concludes that there is as yet no really effective agent. Of the substances available, urethane alone or in conjunction with another agent is the most effective. The steroids are also of value, and although their effect in many cases is to produce some improvement in the anæmia and to induce a sense of well-being in the patient, they have only a limited effect on the disease process. At the Christie Hospital and Holt Radium Institute a relatively small number of cases have been treated with a variety of agents. In approximately one-third of the patients there has been some subjective improvement, usually of a temporary nature, and in an occasional case there has been a prolonged remission. With the agents available there is no statistical evidence that they prolong life. Radiotherapy, therefore, remains the principal method of palliation and chemotherapy should be reserved for the case where radiotherapy has failed to produce palliation.

Radiation Response in Squamous Carcinoma of the Buccal Mucosa.

S. KRISHNAMURTHY (*Clin. Radiol.*, January, 1961) presents an analysis of 124 cases of carcinoma of the buccal mucosa in order to evaluate the various factors which could have influenced the radiation response of the tumours. Overall size modified the response to a certain degree, but the macroscopical tumour type was of much greater importance. Superficial papillary and superficial ulcerating growths responded well to radiotherapy, but infiltrating tumours did not. The tumour bed of the last group showed marked fibrosis and a poor blood supply. Similar changes were present in most of the recurrent cancers, partially explaining their poor response. The grade of malignancy and the duration of the disease had little effect on the response, and there was no evidence that the tumour bed reaction or the age of the patient affected the outcome of treatment.

DNA Thromboembolism after Total Body Irradiation.

Z. HRUBAN *et alii* (*Blood*, November, 1960) reports a fatal case of desoxyribonucleoprotein (DNA) thromboembolism following total body irradiation. The patient was an 11-year-old girl suffering from acute leukæmia. After treatment with steroids and chemotherapeutic agents for some months it became apparent that the leukæmic process was accelerating, and the decision was made to give total body irradiation to be followed by transplantation of bone marrow from the patient's identical twin. On the day of irradiation the patient was acutely ill, with generalized bone pain, purpuric spots and bleeding from both nostrils. During irradiation, which lasted three hours, the patient became progressively more uncomfortable and restless. After irradiation a transfusion of whole blood was commenced and 50 mg. of pethidine was administered. The patient's condition appeared to improve, but about two hours after the completion of radiation therapy she suddenly became dyspnœic and anxious and expired. Autopsy showed that death was due to a sudden and nearly complete obstruction of the pulmonary circulation by capillary thromboemboli composed of DNA; these thromboemboli were also widespread in most of the visceral organs. The authors state that the capillary DNA thromboemboli seen in this patient were quite different from thrombi composed of necrotic cells and fibrinoid material sometimes seen in animals dying of leukæmia; they were rod-shaped masses of homogenous, glassy material which did not stain with periodic acid-Schiff reagent. The authors suggest that DNA thromboembolism is not as rare an event as the lack of reports would indicate, and state that dyspnœa appearing during irradiation of leukæmic patients is probably its main clinical manifestation.

On The Periphery.

ON GOING TO HOSPITAL.

THERE can be no doubt that most of my subsequent trouble was due to my failure to observe, for two days, one simple and obvious fact. But perhaps—as Mr. Kipling used to say—I should begin at the beginning.

Nowadays I am, far too much, a chair-borne troop, and my general condition is softer than it should be. On that particular Saturday morning I had indulged in some exercise of a type that I do not often have; later in the day, the muscles that I had used were inclined to protest, and I felt rather stiff that night, when I went to bed. In the small hours of Sunday morning, something woke me out of a deep sleep, and I made some sudden, unpremeditated movement. With one wild, concerted rush all the devils in Hell suddenly jabbed knives and red-hot pitchforks into my lower lumbar region. I persisted in the movement, and there was another jab. I was thankful to subside back into bed and lie there, extremely still, hoping that "whatever-it-was" would be better by morning.

Of course it was not; and the only thing to do seemed to be to stay in bed. I remained in that particular bed for two days, becoming, if anything, rather worse; and then, suddenly, I saw the light and, in happier circumstances, could have kicked myself for being so dense. Although my bed has quite a firm base, it is fitted with a rubber mattress. Every time one moves on a rubber mattress, one bounces slightly, and on the rebound the mattress comes back and administers a gentle slap, which is exactly the thing to stimulate into further activity muscles that are already on guard. Accordingly I moved to another bed, fitted with a fairly rigid inner-spring mattress. No doubt an orthopædist would say that it is far too comfortable; indeed it was, subsequently, so described; but the fact remains that, from then on, I started to improve.

But by that time, of course, the damage was done. A doctor is not without honour, save in his own household. My family had seen me lying there for more than two days, following my own formula of rest in bed, and now unanimously decided that it was time that something was done; they called in Somebody Who Knows Something About It. The expert arrived on the evening of the third day, having obviously been given the impression that I was far worse than, by that time, I actually was. He brushed aside my suggestion that, in my new bed, I was improving rapidly, and began to talk gravely of the necessity for putting me up in extension, which obviously would mean my transfer to hospital, where, he warned me, I would remain for three weeks.

From that time forth, I had no further say in my own fate. I had a comfortable night, and was able to move quite freely next morning; but the arrangements had been made, and my feeble protest did not avail. At 10 a.m. the ambulance arrived, to take me to hospital. In the good old days an ambulance vehicle was on a chassis with a long wheel-base and fairly big wheels, giving a comfortable ride in most circumstances. Indeed, I remember one, built on a vintage Rolls-Royce chassis, that was almost perfection. But now, unfortunately, the fashion in this part of the world is for a short, stubby, resilient type of vehicle whose motion over the average road resembles that of a yacht beating into a stiff south-wester on Port Phillip Bay.

I am a keen yachtsman and have knocked about a good deal in small boats. In my younger days I was also keen on flying, and for some years I held a private pilot's licence. These are, perhaps, odd hobbies for one who suffers quite easily from travel sickness; but at least I can say that they have given me a wide acquaintance with nauseating situations. In the course of over fifty years' experience, I have never met anything quite so sick-making as a journey of ten miles, in a recumbent position, over a winding road, in a modern ambulance; and I earnestly hope that I never experience it again. I did not actually vomit; but I certainly could not have held on for much longer, and I arrived at the hospital pale and in a lather of sweat. These signs, in turn, were interpreted by the nursing staff as indicating the intense agony that I was suffering as a result of my back injury, and no protestation on my part could disabuse them of this idea. Consequently the expert, once again, received the impression that I was much worse than I actually was. To give him his due, he quickly realized the actual position when he saw me again.

It is nearly thirty years since my previous experience as a hospital patient. Then Mr. (as he then was) Alan Newton had operated on me, and I had spent three weeks in a private hospital in East Melbourne. The whole place ran with quiet, unvarying efficiency, and the most important word in this sentence is "quiet". Doubtless the floors were swept and polished; but my recollection of the process is that it was silent. Doubtless bed-pans and urinals were emptied, and crockery was washed; but there was no noise. Meals appeared without fuss, and they were edible; patients were sponged, and beds were made; somebody looked in from time to time, to see how one was; but there was no noise, and one could sleep.

No so this other institution (which was not situated in East Melbourne), in which I now found myself. Many times I have tried to analyse the various noises, to decide what caused them. Some, of course, were obvious. There was the vacuum cleaner that roared along the corridor, and into and out of each room in turn, gradually approaching in a *crescendo* of din, finally arriving *fortissimo*, and slowly—far too slowly— receding into the distance. There was its mate, the floor-polisher, which approached in similar fashion about half an hour later, with a more subtle whining note that was equally irritating. There was a roar of conversation, that seemed to go on all day and far into the night, as members of the staff shouted to each other up and down the corridor. There was the noise of portable wireless sets, apparently approximately one per patient, each tuned in to a different station, and all adjusted so as to be audible by the deafest man. There was the time when, one after another, no less than seven members of the staff (I never sorted out their exact places in the hierachy) within a space of forty minutes each put a head around the door, asked me if I was all right, and impressed on me that I must push the bell if I wanted anything—each one, in turn, rousing me up again just when I seemed to be dropping off to sleep. Even worse was the young woman who did awaken me, at 5 a.m., to ask whether I wanted milk and sugar in my tea, and her companion, who came in at 5.30 a.m. to take my temperature.

But, as well as all these, there were other noises that defy analysis. In particular there was one, that went on day and night, and sounded exactly like a continuous washing-up of dishes; but surely no hospital in the world finds it necessary to wash dishes, around the clock, in a corridor immediately opposite a patient's room. I give it up. This institution was situated in a fairly quiet street, and therefore the noise of traffic was not troublesome; but had it been on Broadway or Fifth Avenue, the traffic still could not have competed with the noises produced inside the place. Years ago I once spent a night on a bench on the station at Crewe, a busy railway junction on the North-Western route from London to Scotland, famous for the amount of night traffic that it handles. If—which God forbid—I should ever be faced with the choice between spending a similar night at Crewe or in this hospital, I shall choose Crewe; the bench will be just as comfortable as a bed fitted with fracture boards, and the surroundings will be so much quieter.

I only stayed one night in the place. I managed to talk my way out of it next day and went home, where I spent most of the following 48 hours catching up on lost sleep. Yes, I went back to work at the end of the next week.

I am not in general practice. My particular specialty never takes me inside hospitals in the normal course of events, and therefore I know less of what goes on there

than almost any other member of the profession. In my younger days, hospitals were run for the benefit of patients; indeed, as a student, I had it impressed on me, again and again: "The patient is the most important person here." If my recent experience is typical, and I assume that it is, modern hospitals are run to make things easy for the staff. It may be that, in these days of staff shortage, the change is inevitable. But, if that is so, does not the question arise: "Is it a good idea to send a patient to hospital?" This question, I suggest, deserves much more consideration than it usually gets.

"DUNERNE."

A CITY BENEATH A LAKE.

THE following extract is taken from *La Presse médicale* of November 5, 1960, page 1866.

Because of the dredging of the river Gasawka, in northern Poland, the waters of Lake Biskupin fell more than six metres, to reveal the remains of a city enclosed in a double row of fortifications, whose wooden pillars are still in place. Only one entrance is left. The city must have been built on an island, which is now below the waters of the lake because their level has risen over the centuries.

The city dates from about 550 B.C., a time when Mediterranean civilizations believed that the whole of the north was inhabited by barbarians and savages. It must have had about 1500 inhabitants in its rows of identical houses within the fortifications. The various objects discovered are of bronze and iron. Circles, probably sun-symbols, suggest what the religious beliefs of the inhabitants must have been. The city must have had dealings with distant countries, for there were found in it pearl necklets, magic charms and amulets like those found in Egypt, Greece and Persia.

This is a new indication of the trade routes and the communications possible by land and sea in those far-off times.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on October 22, 1960, at the Ballarat and District Base Hospital.

Klinefelter's Syndrome.

DR. D. O'SULLIVAN discussed the case of a man, aged 44 years, who had been referred for advice about weight reduction in the hope of lessening the severity of chronic low-back pain which had made him an invalid pensioner. He seemed an easy-going and placid person, little concerned by the fact that he was a pensioner at his age. On examination, he was seen to be obese, his skin was fine and he lacked body hair. The pubic hair was of female distribution, and axillary hair was sparse. Considerable breast development gave a remarkably feminine appearance to the trunk. The scrotum was small and contained two small soft testicles which lacked testicular sensation. The patient had never had an erection nor had he displayed interest in the opposite sex. A number of investigations were carried out at the Professorial Unit of the Royal Melbourne Hospital. The excretion of 17-hydroxysteroids and 17-ketosteroids was within normal limits. Biological assay of follicle-stimulating hormones gave a result within normal limits. Of the leucocytes, 3·5% contained chromatin bodies of the so-called female pattern. Examination of a buccal smear showed a considerable number of nuclei with chromatin dots. A skin biopsy followed by tissue culture and a chromosome count showed that a majority of cells had 47 chromosomes with an XXY sex chromosome pattern. The result of a testicular biopsy was not available.

Dr. O'Sullivan discussed some recent investigations into the cause of gonadal dysgenesis. In particular he discussed Turner's syndrome, Klinefelter's syndrome, and the testicular feminization syndromes in which the nuclear sex was at variance with the phenotypic or body-build sex.

Difficulties in the Diagnosis of Heart Pain.

DR. DAVID ALEXANDER presented a paper in which he attempted to show the difficulties existing in confirming or rejecting a diagnosis of coronary heart disease in a patient complaining of thoracic or abdominal pain. He also attempted to emphasize how important it was that the diagnosis be made quickly and confidently. Dr. Alexander said that the patient consulted his doctor complaining of pain in the chest or epigastrium possessing certain qualities. The physician examined him and perhaps performed special tests. He formed an opinion regarding the patient's cardiovascular system. The discussion which ensued between the physician and the patient was of the highest importance; it might make a healthy man into an invalid, or it might encourage a man in a precarious state of health to undertake foolish or even fatal physical activity. To illustrate his points, Dr. Alexander presented four clinical histories.

The first patient, a man, aged 38 years, had been admitted to the Ballarat Base Hospital in 1955 suffering from severe central chest pain which had come upon him whilst doing vigorous farm work. He said that for ten years he had had attacks of pain in the epigastrium and the lower part of the chest which, when present, severely limited or prevented physical exertion. A full examination including electrocardiography and X-ray examination with an opaque meal gave normal results. He was reassured and discharged from hospital. During the next nine months he reported as an out-patient five times complaining of anterior chest and epigastric pain, which seemed to spread up through the thorax into the neck. Examinations always gave normal results. He was the subject of severe emotional stress and resentment against his wife. He was discouraged from attending the Ballarat Base Hospital, and went to the Royal Melbourne Hospital. A verbal report indicated that investigations there gave normal results. The patient died suddenly in 1958, a few moments after the onset of chest pain whilst he was doing farm work. A coroner's autopsy indicated that death was due to coronary artery disease.

The second patient was a man, aged 33 years, who had first complained of anterior chest and left elbow pain in 1958. The pain came on whilst he was having lunch, and lasted more than 24 hours. He was examined by a capable cardiologist about one week later. Examination, including an electrocardiography, gave normal results. He continued to have central chest and left elbow pain, sometimes coming with exertion, sometimes wakening him at night, always associated with flatulence, sometimes relieved by antacids, sometimes by trinitrin. When he was examined by Dr. Alexander in 1959, 15 months after the onset, physical examination again revealed no abnormality except for moderate pectus excavatum. A resting electrocardiogram was normal. An exercise test, terminated after the patient had done about one and a half times the standard test because of breathlessness and muscle fatigue, reproduced his pain about four minutes after its completion, when an electrocardiogram was regarded as normal. He was reassured about his heart. About two weeks later he died suddenly whilst walking from an office to his car. A coroner's autopsy indicated that death was due to coronary artery disease.

The third patient, a man, aged 41 years, was examined in 1954, when he said that for two years he had had episodes of pain in the front of his throat from the larynx to the sternal notch. He had been referred to an ear, nose and throat surgeon, who had reported no abnormal findings. He consulted another ear, nose and throat surgeon in Melbourne, who performed a bronchoscopic examination and was said to have removed a small piece of tissue, assuring the patient that he would be relieved of his pain. Another doctor took a complete history (apparently the first occasion on which that had been done), and noted a relationship between the pain and physical exertion. The patient was referred for electrocardiography. A resting tracing was normal. Exercise reproduced his pain, and a tracing was then diagnostic of myocardial ischæmia.

The fourth patient, a man, aged 61 years, was examined in 1953, when he complained of soreness and tightness in the throat, and ear, nose and throat examination revealed no abnormality. In 1955 he wakened with tightness in the throat and chest. A pericardial friction rub was heard, but subsequent full investigation in the hospital revealed no abnormality. At that time, as the result of Dr. Alexander's indecision and the patient's desire for attention, he became convinced that he had heart disease. He then began to complain frequently of chest pain, which came on only at night, never during the day. Electrocardiograms taken at rest and after exercise were normal. A Repatriation Department consultant thought the evidence suggestive of coronary

disease, and the patient was granted a 100% pension. There were aspects of the illness which to Dr. Alexander made that diagnosis untenable. His pension entitlement for coronary disease was withdrawn when it was shown that he had an hiatus hernia and irregular œsophageal peristalsis. He then changed his doctor, and his new physician was persuaded beyond doubt from the history and electrocardiogram that the patient was suffering from coronary disease with heart pain. The Repatriation Commission appointed an independent authority, consisting of a professor of medicine in the University of Melbourne and a member of the Professorial Medical Unit, to review the whole case. The decision was that the patient was not suffering from, nor had ever suffered from, coronary artery disease with angina. However, the patient was by then a confirmed and incapacitated cardiac neurotic. Dr. Alexander said that he thought that it would be an unusually stable personality that could emerge intact from such a period of medical equivocation.

Dr. Alexander commented on the difficulty which confronted physicians, illustrated by the four cases. He said that two young men died, presumably from coronary disease, with the reasssurance of their physicians echoing in their ears. Another man was subjected to unnecessary surgical procedures, and a fourth was converted to neurotic invalidism. Dr. Alexander asked how the diagnosis could be improved. He said that in his opinion that could be achieved only by more accurate and more elaborate history taking. However, William Evans, in 1955, had made the following statement: "Experience has confirmed the unreliability of a patient's description of his pain as a guide to its cause. . . . In more than four fifths of such patients [i.e., ambulatory patients with chest pain] reliance has to be placed on the electrocardiogram as the only means of telling the cause of the symptoms." Evans described, in a number of papers, marked abnormalities in electrocardiograms which he considered to be diagnostic of coronary heart disease. He agreed that very good and accurately recorded electrocardiograms were essential. Dr. Alexander stated his own opinion in the matter in the following terms: (*a*) Such electrocardiograms were not always available; some direct-writing cardiographs had unreliable idiosyncracies. (*b*) There was a real danger of interpreting as abnormal the variation in tracings which might be normal or due to artefacts—for example, those produced by application of the electrodes. (*c*) There were now so many cases recorded of major myocardial infarctions with normal electrocardiograms that one had always to be aware of the fallibility of electric heart records. He suggested that in the diagnosis of cardiac pain the following considerations applied (he was speaking only of patients in whom physical examination revealed no abnormality). (i) About 50% of ambulatory patients sent for diagnosis of chest pain would have electrocardiographic evidence of artery disease, an ischæmic or an infarct pattern being recorded. (ii) Of those with perfectly normal tracings but with pain suggesting cardiac pain, about half would declare themselves as suffering from myocardial ischæmia when the tracing was repeated after exercise. (iii) In the remainder—those who had normal tracings at rest and after exercise—a careful history was the most accurate diagnostic procedure. It was also the most difficult. It required a detailed inquiry into all aspects of the pain in the recognized manner. That need not be elaborated further; but Dr. Alexander stressed that the patient's meaning when he described symptoms and incidents must be obtained with the utmost clarity and accuracy. In addition, he himself found the following points helpful in suggesting the possibility of coronary disease: (i) a history of coronary disease or of sudden death in one or other or both parents; (ii) a history of long-continued emotional stress or nervous tension; (iii) a history of a change in dietary or physical activity habits from those of the parents or from those of earlier life. If certain physical abnormalities were present, the diagnosis became more probable. They were well known—diabetes, obesity, hypertension, aortic stenosis, etc. It was very useful to remember Levine's observation that heart pain was rare in women in the absence of hypertension, unless infarction had occurred. In the present stage of its development, coronary arteriography could not be regarded as a practical diagnostic procedure. With every possible care mistakes would still occur, as the cases presented showed. When real doubt existed, he believed that the physician's attitude should be one of unqualified optimism.

A Pulmonary Puzzle.

Dr. P. Banting discussed the clinical history of a married woman, aged 45 years, who had presented herself when pregnant early in January, 1960. Her estimated date of confinement was May 20, and she gave a history that her first two confinements had been normal, and that she had been delivered of a third child, stillborn, at eight months' gestation five years before. On examination of the patient, apart from the fact that she was overweight (11 st. 4 lb.) and had a varicose long saphenous vein in her right leg, she was seen to be a healthy, middle-aged woman with the fundus at the 20 weeks' position. She was kept on a reducing diet and continued her pregnancy in good health, using an elastic stocking for support of the veins in her right leg. At her last pre-confinement visit on May 24, the fundus was in the term position and the fœtus was presenting by the vertex. The patient's weight was 12 st. 7 lb., her blood pressure was 130/90 mm. of mercury and her urine was free of albumin.

The patient came into labour on May 30, ten days past her estimated date of confinement, and meconium-stained liquor was leaking from her vagina. After six hours of good contractions with no advance, she was delivered by Cæsarean section because of a brow presentation. The baby had a meningomyelocele, and was transferred to the Royal Children's Hospital, Melbourne. The patient's progress was uneventful until the twelfth day. That evening she was sitting in bed talking to her husband when she suddenly complained of severe pain in the right side of her chest, became breathless and turned very pale. Members of the nursing staff were summoned; the patient was white and clammy and her pulse was rapid. Oxygen and morphine (one-quarter of a grain) were administered.

Dr. Banting said that he had seen the patient within half an hour, and by then her colour and general condition were good, although she was still complaining of pain. Apart from slight diminution of air entry at the base of her right lung, her chest was clear. The patient admitted that she had had discomfort in her right leg just behind the knee from the second day after her operation. A presumptive diagnosis of pulmonary embolus was made, and treatment was commenced with anticoagulants and crystalline penicillin. On the following morning she had hæmoptyses, and chest pain was still present. There was diminished air entry at the base of her right lung with a few crepitations, and her temperature was slightly elevated (99·4° F.). On June 15, an X-ray film of her chest was taken with a portable machine; it showed in the right lung gross loss of volume of the lower lobe and a few areas of disc atelectasis in the upper lobe. The right hemidiaphragm was elevated anteriorly. The appearances were consistent with a large embolus. The patient's temperature had risen to 100·4° F. on the previous day, and the antibiotic was changed to tetracycline.

Over the next six days her temperature slowly subsided, but at no stage was it higher than the previous reading. She still continued to be troubled by pain, and dullness to percussion was present at the right lung and air entry was still diminished. No shift of the trachea was present. On June 27, twelve days after the first X-ray examination, a second was made and showed a small right basal effusion, with a most unusual appearance of free air over the apex of the lower lobe of the right lung. However, there was no fluid level. It was thought possible that the abnormality was a bulla in the lung substance of the apex. There were horizontal areas of disc atelectasis in the upper lobe of the right lung. A Bucky film on June 28 showed little alteration in the appearances.

Over the next 12 days the patient seemed much better, but complained of chest pain, particularly at night, and still had a persisting cough that was only slightly productive. Her sputum continued to be blood-stained for one week. The clinical signs remained much as before. On July 7, an X-ray film showed that the left lung was still clear, and in the upper lobe of the right lung there were radiolucent areas in the apex which were held to be a pneumatocele. Another one was now seen anteriorly. The amount of fluid had much increased and was becoming encysted. The right hilar shadow remained low. The bizarre appearances of pneumatoceles and fluid strongly suggested staphylococcal infection. Dr. Banting said that on July 7 he had sought consultant advice, and had shown the films to Mr. J. Clareborough, a thoracic surgeon of Melbourne. Mr. Clareborough considered that the misleading appearance in the apex of the lower lobe of the right lung was being created by an area of atelectasis below and an extension of the associated effusions along the interlobar fissure above. He recommended (i) postural draining and coughing with breathing exercises, and (ii) bronchoscopy, if the appearances were not improved by those measures. The results of that suggested régime were dramatic. After four days' energetic physiotherapy by Mr. P. Rice of Ballarat, a film

was taken which showed that the appearances in the right lung were almost normal, and no abnormality was seen in the left lung.

Dr. Banting said that in retrospect the case was one of (i) pulmonary embolus, with (ii) collapse of the lower lobe of the right lung, and (iii) pleural effusion spreading up the lobar fissure and creating an appearance suggesting pneumatocele in the apex of the lower lobe of the right lung. He had found the case of interest for several reasons. (i) It provided an opportunity of observing the complications of pulmonary embolus. (ii) It indicated that occasionally X-ray appearances could be confusing. (iii) It gave further evidence of the great value of physiotherapy in the management of chest problems. The mother continued to remain well. The baby developed hydrocephalus after preliminary operation closing the defect, and died from intercurrent infection at the age of two months while at the Royal Children's Hospital for review.

A Review of the Mortality of Prostatectomy.

MR. G. R. DAVIDSON said that the aim of all urologists and surgeons concerned with the surgery of the prostate should be target mortality and morbidity zero. The target could, of course, never be fairly struck; but it was interesting to see whether any of their ranging shots had fallen somewhere near their objective. Statistics could be very misleading. In his opinion the only true index was when all deaths due to prostatism in any hospital series or in private practice, whether the patients were subjected to operation or not, were taken into consideration. At the Ballarat Base Hospital, which was not very large, all patients with prostatic disease were referred to the urological department. The figures he intended to present would include all patients who had died after any form of prostatic surgery between 1950 and 1958, but they did not include some patients who probably had died in terminal retention of urine associated with other illnesses who had not been referred to his department. He regretted that the development of the hospital records did not permit him to give the complete picture in that direction. Private cases which were also shown in the series to bring the number up to a more reasonable total were, he thought, accurate in that respect. Mr. Davidson said that from a clinical point of view there were three types of bladder-neck obstruction requiring operation: (i) that causing subacute or partial obstruction; (ii) that causing acute retention of urine; (iii) that causing chronic retention of urine, often with overflow and infection. There was little argument about principles underlying the treatment of the first and second groups. In the first group time was usually not a factor. The patient could be adequately investigated and prepared, and mortality should be low. The second group of patients were best treated by immediate operation. However, he considered that the word immediate should on occasions be modified by circumstances. Often a brief period of delay allowed a fuller assessment of the general state of the patient. If there was any doubt about the availability of the best possible laboratory and nursing facilities, he thought it wiser to relieve the obstruction by the insertion of a catheter and to operate as soon as adequate facilities could be obtained. Differences of opinion existed as to how the group suffering from chronic retention of urine should be treated. The work of Wilson Hey, Wells and others had shown that many patients suffering from chronic retention of urine could be safely operated on without preliminary drainage. However, the followers of that school of thought said in effect: "If we drain these poor-risk cases in any way, we run a grave risk of introducing an infection which will turn the tables against them and in fact may probably be the end of them. We must in carrying out this policy be prepared to accept a definite mortality rate. We accept this because in the long run we think our results will be better." Mr. Davidson said that he had a definite feeling that catheter infection occurred more frequently and more severely in old hospitals than in new hospitals, in large wards than in small wards, and in some hands than in other hands. There should be less trouble in a community where antibiotics had not been abused, if there was such a community. He admitted that any evidence he had in support of those contentions was purely circumstantial—not so strong as, for example, finding a trout in the middle of the brook, but still strong. If there was any truth in his opinion, surely they should continue in their efforts to control the introduction of infection into the bladder rather than carry out a method of treatment that was associated with an inevitable and accepted mortality rate. Mr. Davidson went on to say that, in common with many of those present, he had been trained in the belief that a bladder-neck obstruction should not be removed surgically until some sort of stabilization of renal function had been achieved. That had been brought about by either short-term drainage with a catheter or long-term drainage by suprapubic cystostomy. As far as the possibility of uræmia was concerned, he was now sure that that attitude was in general wrong. Post-operative uræmia, in his experience, was an extremely rare complication, and when it did occur he thought it was nearly always just one manifestation of a general cardio-vascular break-up. The old men who developed that complication were usually in a very precarious condition, worn out with loss of sleep and dehydration, nitrogen retention, infection and persistent discomfort; they were barely able to stand a haircut and shave unless it was done very carefully in two stages, let alone a major operation such as prostatectomy. Preliminary drainage, as well as improving renal function, allowed the patients to be hydrated and fed and, above all, given an opportunity to sleep. Of course, all surgery consisted in the assessment and taking of calculated risk. He thought that in his hands better results were obtained by some form of preliminary drainage in some of those poor-risk patients with chronic retention. While he was on the subject of risks, it was interesting to note that the mortality of the Wilson Hey operation had been reviewed by Aiken in 1953 in *The British Journal of Urology*. He had given the overall mortality rate as 10%. Mr. Davidson then quoted the figures from the Ballarat Hospital from 1950 to 1958 in the following terms: total admissions to urology department, 236; suprapublic cystostomies, 25; deaths, 5; mortality rate, 20%; two patients discharged with permanent suprapubic tubes; subjected to prostatectomy, 229; deaths, 10; mortality rate, 5%; total mortality rate of all admissions, 6·4%. In the period from 1950 to 1959, there had been 352 private patients; 20 had undergone suprapublic cystostomy and two had died. One had been discharged from hospital with a permanent suprapublic tube; two patients had died with bladder-neck obstruction, a complicating factor, and no operation was performed. In the same period, 1950 to 1959, 349 private patients had been subjected to prostatectomy and eight had died, the mortality rate being 2·3%. The total mortality rate of all admissions was 3·4%. The average ages at death were: public hospital series, 78 years; private hospital series, 72 years. Mr. Davidson went on to say that it might well be asked what, if anything, could be learnt by presenting series of statistics such as he had shown. Perhaps not very much could be learnt; but he thought that it did at least show that it was possible to carry out that sort of surgery in a small place and produce results which were at least comparable with those produced in larger centres. In that field he considered that the provincial city offered many advantages. In general there were probably fewer bad-risk patients, although in all truth some were bad enough. Hospital accommodation was usually easier to obtain, and rarely if ever did a patient have to wait for a bed. With the possible exception of endoscopic resection, prostatic surgery did not demand virtuosity, but it did demand great patience and constant attention. For example, clot retention, unless quickly relieved, could in an elderly patient produce a disastrous and often irreversible series of changes. Sometimes three or more men suffering from prostatectomy would all have hæmorrhages at the same time, and that would produce a state of affairs which he could best describe as a bloody symphony of discordant confusion appreciated only by the macabre, mainly, he supposed, because of the gross overtones of the tolling bell. Those patients needed immediate care and personal attention, and the situation became almost impossible if one had to travel miles to give it. In passing, he would like to suggest that there was probably a reason for that sort of thing, and he refused to believe that such incidents were purely coincidental. A common but not understood factor was probably operating—infection perhaps, barometric changes probably. He asked whether some commonly used drug could, under certain conditions, interfere with the synthesis of vitamin K in the patient's own bowel. Perhaps they would know one day. However, there was one thing of which he was certain—there were really only two important factors causing prostatic deaths, hæmorrhage and infection. Most other complications were usually secondary to those, and were often terminal. Of course, age was important. Over the age of 80 years the mortality rate rose rapidly. If the members of the medical profession would take more notice of the warning signs and symptoms of approaching bladder-neck obstruction in the male and persuade him to have treatment before the inevitable disintegration occurred, lives would be saved. There were certain words in the English language which could be described as "weasel" words. They sucked the life blood out of, they overpowered, other words in their immediate vicinity. In medicine the

word old was one of those. "An old man with frequency"; how often did one hear it The frequency was forgotten, they almost forgot that he was a man. He was just old. Mr. Davidson held that the general study of geriatrics would need to have wider vocation, scope and appeal, because there was little point in trying to lengthen a man's life if he felt that he had not much to live for. That might well be the key to the differences found in the average ages of men who died after prostatic surgery in public hospitals and in private practice. Successful prostatic surgery might be said to depend on the control of a host of abnormal factors and states, often synergistic. In that control many people played a part—the physician, the surgeon, the anæsthetist, the biochemist and, last but certainly not least, the nursing staff. The physician had an important part to play, especially in pre-operative assessment of the poor-risk patient. Unfortunately, not even the best of them could answer the impossible question, which was: "with this man's present cardio-vascular and renal status, how long will he live if the bladder-neck obstruction is successfully relieved?" What the physician could say, and usually did say, was that the patient was not going to live at all if the condition was not relieved, and that he probably would not live if it was. The control of operative and post-operative hæmorrhage was essentially the problem of the surgeon. Whatever operation he performed, he must leave a clean prostatic cavity, because only a clean prostatic cavity could contract. The anæsthetist played a vital part. Mr. Davidson liked one who would accept Pope's advice, and be not the first by whom the new were tried, nor yet the last to lay the old aside. He had nothing but the highest respect and regard for those who had worked and no doubt at times suffered with him. The conscientious resident medical officer and the experienced ward sister were important members of the team. When changes occurred there, almost inevitably the results of operation altered also. It was indeed a depressing thought that the physical experience was generally so expensive. Mr. Davidson said in conclusion that he believed that even in the absence of any important new development there were still some factors which, if controlled, would make the surgery of the prostate safer, would make their results better and would allow at least some of their shots to drop nearer the target he had mentioned.

(To be continued.)

Out of the Past.

A BOOK REVIEW.[1]

[From the *Australasian Medical Gazette*, April 21, 1902.]

SEXUAL INVERSION. By Havelock Ellis, L.S.R. (England). Sold only to physicians, lawyers, advanced teachers and scientists. Philadelphia, Pa.: F. A. Davis Co.

THIS work deals with a subject which may be of some interest to alienists and of some assistance to medical jurists, and that is about all that can be said of it. A record of a number of cases of sexual inversion in the two sexes, with a description of their experiences in various abnormal ways of gratifying the sexual appetite is not edifying reading to any pure-minded person, whether he be physician or lawyer or scientist. To speak of such a work as "scientific" is, in our opinion, to use a misnomer.

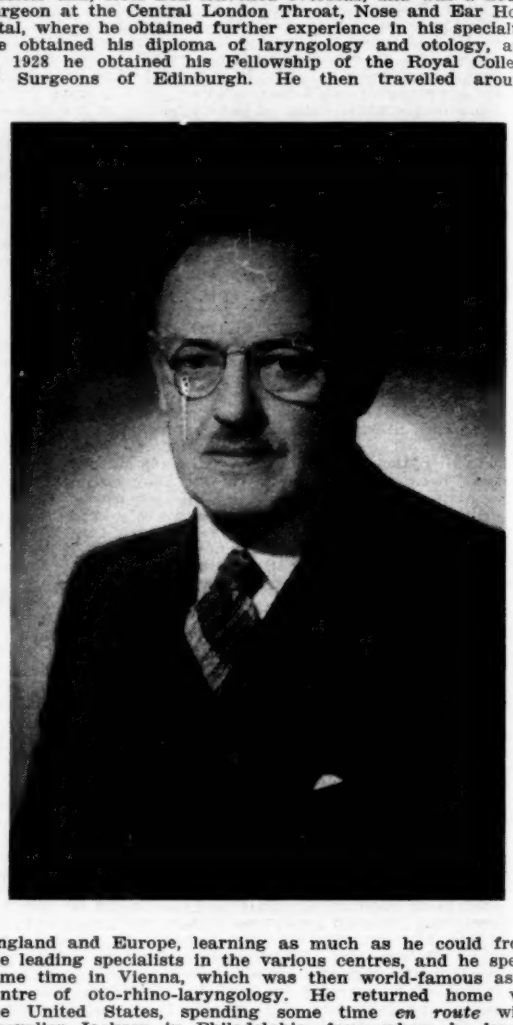
Obituary.

NOEL EDWARD HAMILTON BOX.

WE are indebted to DR. PETER FREEMAN for the following account of the career of the late Dr. Noel Box.

Dr. Noel Edward Hamilton Box, who died, aged 58 years, on May 13 last, was the second son of the late Dr. John Box, one of the earlier eye, ear, nose and throat specialists of Melbourne. Noel Box received his earlier education at Scotch College, Melbourne, and later at Geelong College, where he was a member of the first eighteen, and took a most active interest in boxing. He commenced his medical course at the University of Melbourne in 1920, and after graduating in 1925, he was a house surgeon at the Victorian Eye and Ear Hospital for the following two years. Subsequently, he was a *locum tenens* for Dr. Stewart Hills, an eye, ear, nose and throat specialist of Ballarat, for a year.

After this, Noel Box travelled overseas, and was a house surgeon at the Central London Throat, Nose and Ear Hospital, where he obtained further experience in his specialty. He obtained his diploma of laryngology and otology, and in 1928 he obtained his Fellowship of the Royal College of Surgeons of Edinburgh. He then travelled around



England and Europe, learning as much as he could from the leading specialists in the various centres, and he spent some time in Vienna, which was then world-famous as a centre of oto-rhino-laryngology. He returned home via the United States, spending some time *en route* with Chevalier Jackson in Philadelphia, from whom he learnt many of the techniques of endoscopy that he ably used throughout his life.

On his return to Melbourne, Noel Box devoted much of his time to public hospital work. In 1930, he was appointed honorary assistant surgeon to the Ear, Nose and Throat department of the Alfred Hospital, and honorary assistant aural surgeon to the Victorian Eye and Ear Hospital. He was a clinical assistant to the Children's Hospital for a number of years, and also ear, nose and throat consultant to Fairfield Infectious Diseases Hospital for a period. He was admitted to Fellowship of the Royal Australasian College of Surgeons in 1932. In 1936, he was promoted to the position of honorary aural surgeon to the Victorian Eye and Ear Hospital, from which position he resigned at the commencement of the second World War. In 1946, he was appointed honorary ear, nose and throat surgeon to the Alfred Hospital, and on his retirement in 1956 he was appointed honorary consultant ear, nose and throat surgeon. He was also consultant ear, nose and throat specialist to the Williamstown and District Hospital for many years.

Noel Box gave much valuable service to the Royal Australian Air Force, especially during the war. He attained

[1] From the original in the Mitchell Library, Sydney.

the rank of group captain, and wes senior ear, nose and throat consultant on R.A.A.F. headquarters staff for many years, a post that he held at the time of his death. He was a member of the board of examiners for the diploma of laryngology and otology in the University of Melbourne, and he exerted considerable influence in the teaching of ear, nose and throat diseases in this city. He was a member of the Melbourne Cricket Club, and of the Naval and Military Club.

Noel Box was always mechanically minded, and when he first returned from overseas and had more leisure, he made many instruments which he used throughout most of his life in an extremely busy practice. Everything which he undertook he did in the most thorough and painstaking manner. Time was never any worry to him, and he would never allow himself to be hurried over any particular problem. However pressing his personal arrangements might be, he would not allow himself to be hurried into making a cursory examination of a patient, or a diagnosis which was not well founded by very sound reasons. He always maintained a keen interest in his patients, and never forgot that his patients were individuals in need of his help. His surgical technique was most meticulous and thorough, and he did not leave anything until it had been performed to his entire satisfaction. Much of his success can be attributed to his painstaking surgical technique, and the rewards of this are illustrated by the large numbers of his grateful patients.

His greatest hobby was gardening, and he had a wonderful store of knowledge on this subject. He was the owner of a large garden filled with rare trees and shrubs. Rhododendrons were his delight, and he imported many varieties from New Zealand, England and parts of Asia. During the last ten years of his life he turned his flair for horticulture to farming, on a property at Lower Tarwin, which he greatly improved by experimenting with trace elements and different grasses for pastures. He turned 2800 acres of useless country into good pasture of heavy carrying capacity. He established a fine library on agricultural lines, and he seemed to know to which page to refer for any information on any subject under discussion.

During his very active life, he built up a wide circle of friends, and he had a very large private practice. His sudden death came as a great shock to his patients as well as to his family and friends, and his passing leaves a gap which will be hard to fill. He is survived by his widow, two sons and a daughter, to whom we extend our deepest sympathy.

DR. A. R. HAYWOOD writes: To have known Noel Box and to have had the privilege of his friendship and guidance over many years is an experience never to be forgotten. Whatever he undertook was carried out with care, thought and skill. His sporting activities in the field were as carefully planned as his medical procedures. In his later days he bought a large area of unimproved country and developed from it a productive property. This was characteristic of his outstanding capabilities.

During my association with him at the Alfred Hospital, I was always impressed by the way he would spend an indefinite time on a case until he was satisfied that he understood it completely. He took great pains to explain to each patient the nature, treatment and possible course of his or her complaint. Noel Box always said that mistakes were not made by lack of training and knowledge, but by not schooling oneself to take time in examination and investigation.

His kindly treatment of patients, both in the public hospitals and in private practice, was phenomenal. His patients have said to me: "He seemed more like a friend than a specialist."

One could write indefinitely of his skills and accomplishments, but let me conclude with these words—it was an honour to know Noel Box, and we should be better men for having done so.

AIR VICE-MARSHAL E. A. DALEY writes: Noel Box was first appointed to the R.A.A.F. as a Citizen Air Force specialist in 1936. From then on he showed great interest in the ear, nose and throat problems of aviation, and this grew with the onset of war and the increase in complexity of environmental situations which affected both aircrew and ground staff. For the first few years of the war he acted as ear, nose and throat specialist to the R.A.A.F. in Melbourne, and sometimes made visits to other States. His work involved, not only consultations, but surgery, at Laverton and later at No. 6 R.A.A.F. Hospital, Heidelberg. He was an inspiration in his field to younger medical officers, and his opinions were eagerly sought by colleagues in associated specialties.

Although his contribution to the civil community and the R.A.A.F. over these early war years was great, he himself was not fully satisfied with it as such, and in early 1944 he was attached, for full-time duty, to Air Force Headquarters, and was almost immediately sent to New Guinea, to act in a specialist capacity and to make a particular study of tropical otitis externa. His report on this was used as an instructional memo from the Director-General of Medical Services for unit and other medical officers. He resumed his specialist part-time duties later in the year. However, once more he returned to the active list for full-time duties in early 1945, and served in the islands and in association with No. 5 Medical Receiving Station until his demobilization in November of that year.

In the post-war years, the introduction of jet aircraft with their high rates of ascent and descent and their problems of high-intensity noise created a field of great interest to him, which he followed up, and in which he made specific personal contributions for the protection of aircrew and ground staff in these new environmental situations. His article on barotrauma in 1952 was of considerable value to all concerned. Although always extremely busy, he made a special trip to the Australasian Medical Congress (B.M.A.) in Hobart in 1958 to take part in the Services section discussion of high-intensity noise. In association with the Commonwealth Acoustic Laboratories, he assisted, from the professional point of view, the introduction of the R.A.A.F. hearing conversation programme at jet bases. Up until his last few weeks he was actively engaged also on the ear, nose and throat advisory panel of the Medical Services Committee in Defence as the R.A.A.F. representative. He held the rank of group captain, and his passing is a great loss to the R.A.A.F., in which he was held in the highest esteem by all, not only for his professional contributions, but also for his own fine personal qualities.

Correspondence.

STORAGE AND SUPPLY OF IMMUNIZING MATERIAL.

SIR: Through your columns I would like to address some observations and questions to Dr. Bazeley of the Commonwealth Serum Laboratories, and I hope he will reply through your columns so that many others like myself will be put out of the confusion which has arisen over poliomyelitis and tetanus immunization.

I do not blame Commonwealth Serum Laboratories for the present shambles and chaos of the polio campaign, or for the complete failure of the quadruple antigen arrangements whereby the immunization campaign was advertised to commence on February 1, and apart from a handful of doses, the vaccine is still not available to date. I would suggest, however, that if the authorities concerned put their minds to the problem of distributing the vaccine, both polio and quadruple antigen, through the general practitioners instead of through local government bodies, a more satisfactory state of affairs would exist.

The main reason for the present form of distribution is stated to be the lack of refrigeration facilities and the essential need to refrigerate the vaccine. However, in October it is reported that Dr. Bazeley emphasized the serious consequences of inadequate storage facilities for vaccine. Only 23% of chemists have a refrigerator in the pharmacy. Tetanus toxoid is as critical as poliomyelitis vaccine as far as storage is concerned. I would therefore ask:

1. Is it a fact that tetanus toxoid is just as susceptible to deterioration as polio vaccine?

2. If so, why has this fact not been stated and clearly emphasized to the profession, apart from the small reference to storage tucked away, in small print, on the enclosed literature?

3. If tetanus toxoid is similarly prone to deterioration as polio vaccine, why not a national campaign through local councils as for polio? Surely the mortality rate in tetanus must far exceed that of polio (except perhaps under severe epidemic conditions).

4. In my suburb of five chemists, not one possesses a refrigerator. When the matter was discussed, the chemists

hastened to point out that the tetanus toxoid was not refrigerated at any of the wholesale distributors and may well spend a whole day in a delivery van in the hot sun. What point in the chemist possessing a refrigerator? In view of this, of the many thousands of people already immunized with tetanus toxoid, how many can now be assured of immunity?

5. If the chemists are not permitted to distribute polio vaccine because of lack of refrigerators, should not Commonwealth Serum Laboratories refuse them permission likewise to handle tetanus toxoid?

6. Finally, what should I do now when I am asked to immunize a patient against tetanus and from whom should I obtain my supplies of tetanus toxoid?

Yours, etc.,

80 Penshurst Street,
Penshurst,
New South Wales.
February 28, 1961.

LIONEL L. WILSON.

SIR: I am pleased to have the opportunity of replying to Dr. L. L. Wilson's letter of February 28, 1961, on the distribution of prophylactics.

The system of distribution through local health authorities was recommended by the National Health and Medical Research Council on the advice of representations of the medical profession to meet certain fundamental requirements of storage and record not otherwise assured.

We have had numerous complaints in connexion with incorrect storage, and it surprises us that some pharmaceutical chemists carelessly disregard the recommendations. By virtue of their training, they should know that most biological products are inherently labile, and any pharmacopœia or other appropriate handbook will remind them of this fact. "The British Pharmacopœia", "The British Pharmaceutical Codex" and "The Extra Pharmacopœia" refer to the storage of the vaccines and antisera and antitoxins in the following or similar terms:

Antitoxins (B.P.). Antitoxins should be protected from light. Liquid preparations should be kept at temperatures between 0° and 5° C. Solid preparations should be kept at a temperature not greater than 20° C.

Vaccines (B.P.C.). Bacterial vaccines, bacterial toxoids and bacterial toxins should be stored in the dark at as low a temperature as possible above 0° C. They deteriorate at temperatures approaching 20° C.

Again, *The Australian Therapeutic Substances Act 10* (1) states as follows:

> Where a pharmaceutical benefit is a controlled therapeutic substance, a person shall not supply goods as that pharmaceutical benefit under a law of the Commonwealth relating to the provision of pharmaceutical benefits unless the goods conform to the standard for that controlled therapeutic substance.

Elsewhere the Act and Regulations clearly state that products supplied as pharmaceutical benefits must comply with the specifications of "The British Pharmacopœia" or "The British Pharmaceutical Codex", and these authorities in turn clearly lay down the storage conditions necessary to maintain the potency of such products.

We recognize that during periods of transport, it is not practical, except in special circumstances to maintain the recommended temperature conditions, and with this in mind we have allowed for a margin of safety during the manufacture to cover the normal exposure to atmospheric temperatures during short periods of transport. When particularly labile products are dispatched to distributors, or when the period of transport is of long duration in the case of less labile products, we arrange refrigerated transport from these Laboratories. Wherever possible, distribution to our depots is made by air transport. Therefore we feel certain that the many persons already immunized with tetanus toxoid can be assured of immunity, provided the preparation is not kept for long periods under adverse conditions.

Practitioners should impress on their pharmacists the necessity to provide refrigerated storage for biologicals. This matter has been discussed with the Pharmaceutical Guild, and with their cooperation, it is hoped that in future these facilities will be more readily available. However, I am sure Dr. Wilson will appreciate that these Laboratories cannot maintain constant supervision over the storage of biological products in the wholesale houses and pharmacies to which they are distributed.

Yours, etc.,

Commonwealth Serum Laboratories,
Parkville,
Victoria.
March 21, 1961.

P. L. BAZELEY,
Director.

RESEARCH BY THE GENERAL PRACTITIONER.

SIR: As the subject of research is mentioned from time to time, I am itemizing some of the research work which I have done as a medical officer in hospitals and as a general practitioner.

I described a method of preventing vomiting in patients under general anæsthesia. Published in THE MEDICAL JOURNAL OF AUSTRALIA, the method was confirmed in Canada.

I grew *Penicillium* mould 20 years ago on a liquid medium. I found that, applied to the skin lesion, the mould cured chronic non-healing ulcers and intractable tinea following a single application. (The active principle which cured the tinea is now available in tablet form as griseofulvin.) I found that maintenance injection of pituitary hormone cured alopecia (published in THE MEDICAL JOURNAL OF AUSTRALIA as a letter).

Following the use of injections of a blue dye used to measure vitamin C deficiency, I noted that if adrenaline 1:1000 solution was injected in the area at the same time, the dye was not decolorized until some two hours had passed. Without the adrenaline the decolorization occurred in two to ten minutes. This observation was the basis of research I suggested which led to the knowledge that adrenaline solution, injected into an area bitten by a venomous snake, delayed absorption of the venom for two hours. The venom, however, was not destroyed.

In a case when a man was bitten by a venomous snake, presumed to be a tiger snake, I made detailed observations. He had antivenene injections. He required artificial respiration for two hours. The tourniquet could not be removed without worsening until four hours had elapsed.

I found the first four cases of phenylpyruvic disease (phenylketonuria) in Australia, and carried out various investigations. These cases were reported in THE MEDICAL JOURNAL OF AUSTRALIA.

Using samples of the drug "Dilantin" before it became available commercially, I noted that epileptics treated with the drug required extra vitamin C. The same fact was ascertained simultaneously by a research worker in the United States of America.

In New Guinea I noted that conditions which in the white man would be highly malignant were not malignant in natives. These observations were confirmed by the pathologist, and some years afterwards they were confirmed by a medical officer in New Guinea, whose article was published in THE MEDICAL JOURNAL OF AUSTRALIA.

I also noted the condition of salivary cells in heavy smokers. In non-smokers the cells, fixed and stained, were normal. In the heavy smoker group the cytoplasm was less clear with less detail; while the nucleus of every cell was thickened and deeply stained. The condition in such cases could be described as early precancerous. Presumably in most cases no further change occurred, but the same changes were noted in the saliva of patients suffering from bronchogenic carcinoma.

In an article of mine published in THE MEDICAL JOURNAL OF AUSTRALIA, I noted that mongolism was hereditary and I quoted the instance of three mongoloid children occurring in one family. This was the first instance of the kind in the literature of the British Commonwealth. A similar finding was made by a medical practitioner in England some years later; data concerning this will be found in the *British Medical Journal* in a number published about eight years ago.

After noting Brissaud's reflex in alcoholic cases, I found two new reflexes in chronic alcoholics. The muscular response in both varieties of the new reflexes was movement of the arm muscles as in swimming (at the stage of evolution of the crocodile). The reflexes were elicited in some cases by tapping the infrapatellar tendon, and in others by stroking the sole. The reflexes are evanescent, elicitable

few times, but again elicitable later. It is to be noted that the primitive channels used by such reflexes may be turned to good use. Phelps' method of obtaining movement in spastic children is an example.

I would like to add that most of the above research work was carried out without any but the simplest equipment.

Yours, etc.,

S. J. CANTOR.

"Nalabar",
Woolsthorpe,
Victoria.
March 14, 1961.

PRESCRIPTION FORMS.

SIR: The decision of the Government not to print any further prescription forms for "free medicine" illustrates how slowly requests made to Government departments are dealt with and how conditions change. The original request was made to a Labour Government and the official B.M.A. reasons then for not using Government forms were as follows:

The Government refuses to accept a doctor's prescription on his own prescription forms as entitling a patient to free medicine, but insists that all prescriptions be written in duplicate on a Government form. I refuse to carry out this procedure for these reasons: (*a*) It is entirely unnecessary. (*b*) It would require me to spend a considerable part of my valuable time performing clerical work that could be done by others. (*c*) It would immediately bring me under Government control, and I am not prepared to run the risk of being fined £50 or being sent to gaol for three months for some so-called "offence" against the Act or the Regulations under it.

The position at present is that prescriptions on the "free list" are used by practically all members of the profession (and written in duplicate). The penalty for prescribing drugs on the restricted list without observing the regulations may be well in excess of £50, as the doctor pays for all drugs so used; and this applies equally to drugs prescribed on his own prescription forms as well as to those provided by the Commonwealth.

As a constant but rather clumsy user of the Government forms over a number of years, may I say that they appear to be no more dangerous to have in one's possession than telegram forms; that they are smaller and much handier to use than Repatriation forms; and that the carbon shows up better on the duplicate. All the original objections to the National Health Service forms and procedure have been swallowed, and no objection raised to the Repatriation forms (which are in triplicate and equally free).

I can only hope that this concession on the part of the Government to a B.M.A. request of many years ago is not a prelude to concessions by the B.M.A. towards the further socialization of the profession as a *quid pro quo*.

Yours, etc.,

T. LOVEGROVE.

Wongan Hills,
Western Australia.
March 10, 1961.

HYPERTENSION AND LIFE ASSURANCE.

SIR: Though Dr. Woolnough (MEDICAL JOURNAL OF AUSTRALIA, March 11, 1961) has set me the task of handling editorial forbearance, I appreciate his courtesy in giving me the opportunity of saying that no opprobium was implied. The terms "fudge and fabricate", to which, maybe, he takes exception, and which, incidentally, were governed by the first personal pronoun plural, were meant, in so far as "fudge", much in the sense that we chided one another during games of marbles and leap-frog; the choice of the word "fabricate" is explained in a preceding paragraph, the authority for which is expressed as "it is generally believed".

The other quotation on which Dr. Woolnough has asked me to enlarge, i.e., "Pending such time when an inexpensive and objective method of recording the blood pressure is devised, the task might be better left to technicians"—relates to words inadvertently omitted by Dr. Woolnough, e.g., "influenced by a knowledge of the vagaries (i.e., in blood pressure readings) we know to exist or because of lack of surety in recording". In explanation, no one endures greater travail than the writer in being sure, when examining some applicants for life assurance, just what is the casual or basal blood pressure or how labile it is, hence the idea of the "objective" or graphic recording instruments; a technician may often be more patient and appear as less of an ogre to a person than a doctor, and thus likely to secure a more helpful record.

For the rest, might I add that I regard blood pressure taking as an art too little regarded; that attention to factors such as armlets, manometric faults, a savour of exactness with figures ending in units as well as in "0", is essential if we are to have worthwhile statistical studies and ensure equity in life risk assessment.

Yours, etc.,

W. J. MCCRISTAL.

The City Mutual Life Asurance Society Limited,
Hunter and Bligh Streets,
Sydney.
March 16, 1961.

A CASE OF MISTAKEN DIAGNOSIS.

SIR: Two comments have been made about my letter (MED. J. AUST., February 4, 1961).

Firstly, I would like to reply to Dr. Mark Harrison (MED. J. AUST., 1961, 1: 187). He suggested that the correct diagnosis should be congenital microcoria (miosis). He may be correct. I must admit that I did not think of this; but is miosis necessarily related to progressive diminution of vision, as tobacco-alcohol is? There was no indication of any other sign of Marfan's syndrome, such as arachnodactyly. It is possible that the correct diagnosis might be both. It was certainly not glaucoma, so its remains a case of mistaken diagnosis. even if that involves me too. I am, however, grateful to Dr. Mark Harrison for his intelligent suggestion.

Secondly, I would like to reply to Mr. F. Harold (MED. J. AUST., March 4, 1961). He suggested that my views about the arsenical content of beer were unreasonable, and in the light of his authoritative statistics, I give way to him. In this, I do not give way to him. He states: "Surely it is an established fact that alcohol is neither a primary nor an accessory factor in the destruction of vitamin B_1."

Starting from page 3009 of Sir Stewart Duke-Elder's "Text Book of Ophthalmology", Volume 3, under the heading of "Toxic Amblyopia", the initial statement is: "Tobacco Amblyopia—or as the condition is sometimes referred to, Tobacco-Alcohol Amblyopia, because of the usual adjuvant effect of the latter poison—is the most common type of toxic amblyopia met with in England." Sir Stewart goes on to say, in succeeding pages, that while the two poisons probably act in conjunction, there is no doubt that amblyopia can result from tobacco alone, uncomplicated by alcoholism, but that in either case the treatment is the administration of vitamin B_1, to replenish the destruction caused by either or both poisons. I am, however, obliged to Mr. Harold, for correcting me in my ideas about the arsenical content of beer.

Yours, etc.,

ARTHUR D'OMBRAIN.

135 Macquarie Street,
Sydney.
March 14, 1961.

INFECTIOUS HEPATITIS.

SIR: In your issue of February 11, 1961, Professor C. R. B. Blackburn contributes an informative article on infectious hepatitis, in the general series "Brush Up Your Medicine". I am sure many will agree that it is valuable and timely to have such a clear exposition of the clinical aspects of this all too prevalent disease. It was, however, with some surprise that I read the statement: "The virus hase been isolated from the serum and fæces of patients in the acute phase of the disease and has been demonstrated to be present in convalescent serum for months."

The presence of virus in convalescent serum is reasonably inferred from transmission experiments on human volunteers, but this is surely a very different matter from isolation. I would be interested to learn of success in the isolation of hepatitis virus by tissue culture or other techniques. A specific laboratory test for infectious hepatitis would be of the greatest value. The isolation of the virus would surely be as great a contribution here as the isolation of

poliomyelitis virus has proved in that disease. It would also open up the possibility of successful protective immunization with inactivated or attenuated virus vaccine.

In the meantime, it would be a pity if the statement quoted were to lead to numbers of requests for hepatitis virus isolation as a routine diagnostic measure, or to questions as to why a vaccine had not yet been produced.

Yours, etc.,

169 Rundle Street, Adelaide.
P. S. WOODRUFF.

February 17, 1961.

SOME FACTORS IN THE PREVENTION OF SEPSIS IN OPHTHALMIC SURGERY.

SIR: The paper by Dr. D. O. Crompton, "Some Factors in the Prevention of Sepsis in Ophthalmic Surgery" (MED. J. AUST., 1961, 1: 356) describes further disastrous results that can occur to eyes and vision by the use of contaminated eye-drops. The cases cited confirm other reports in ophthalmic journals. Dr. Crompton shows the dangers of corked bottles and of introducing soiled droppers into eyedrops.

It is deplorable that the Commonwealth Health Department, through instructions covering the supply of Pharmaceutical Benefits, imposes such dangers upon the users of extemporaneously prepared eye-drops because the cost permitted does not include the small additional charge required for the provision of eye-drop bottles. If dropper bottles are not issued, many people will use droppers that have served previously for other eye drops, for administering ear drops or for measuring medicines, or even those used by other members in the family or factory.

As well as the direct dangers from contamination, other effects occur. Accelerated deterioration of the active medicament occurs in infected eye drops. This is very important where the medicament is in low concentration (e.g., the alkaloidal salts of pilocarpine and atropine). The sight-saving properties of these drugs can be lost by small reductions of potency.

The extemporaneously prepared eye drops of the National Health Service are dangerous and out of date. Improvement could be achieved by the provision of eye-drop bottles. Bottles containing eye-droppers should now be provided; but they, in turn, should be replaced by plastic drop bottles as these become more readily available. The position will only be satisfactory when the extemporaneous preparation of commonly used eye-drops is discontinued and manufacturers supply standard, sterile, preserved eye-drops in plastic drop-bottles.

Yours, etc.,

82 Collins Street, Melbourne.
RONALD LOWE.

March 15, 1961.

Public Health.

FOURTEENTH WORLD HEALTH ASSEMBLY.

THE Fourteenth World Health Assembly opened in New Delhi on February 7 and ended on February 24, 1961. The President was Sir Arcot Lakshmanaswami Mudaliar, Vice-Chancellor of Madras University and chief delegate for India. The Assembly was attended by delegates from WHO's 109 member and associate member States, including those from one new member, the Islamic Republic of Mauritania, and from two new associate members, Tanganyika and Ruanda Urundi.

The Assembly adopted a programme of work for 1962, with a working budget of 23,607,180 dollars. This figure comprises the Director-General's original proposal of 20,852,000 dollars, *plus* several additions approved by the Assembly, including an amount of 2,000,000 dollars towards financing the field activities of the world-wide malaria eradication programme, 797,347 dollars for the administrative and operational services cost of the malaria eradication programme, 110,033 dollars for assistance to the Republic of the Congo (Leopoldville), and 21,400 dollars for the use of Russian as a working language in the European region. A deduction of 203,000 dollars was made by the Director-General from his original proposal, following the decision of the General Assembly of the United Nations to reimburse WHO for the book value of its investment in the Palais des Nations in Geneva, WHO's present headquarters. A new headquarters for WHO is at present being built in Geneva.

The budget adopted for 1962 reflects attempts to preserve the continuity of WHO's evolution in recent years, with particular reference to the increasing importance of the stimulation and coordination of medical research, the continued need to concentrate efforts on the world-wide eradication of malaria, the efforts to strengthen basic public health services, and the wish to accept fully the responsibility which a very substantial increase in membership places upon the Organization.

Twelve countries were elected to designate a person to serve on the WHO Executive Board. They are: Chile, Iceland, Iraq, Israel, Italy, Japan, Nigeria, Pakistan, Poland, Senegal, Spain and the United States of America. The twelve other members of the Board are from the following countries: Argentina, Ghana, Ireland, Jordan, Korea, Luxembourg, Nepal, Peru, Sudan, Thailand, United Kingdom and Venezuela.

Among the topics which received particular attention were the following:

Assistance to Newly Independent States.—In its penultimate session the Assembly unanimously adopted a resolution concerning the granting of independence to colonial countries and people. Welcoming the attainment of independence by new States and their entry into WHO, and stressing that WHO has an important part to play in promoting the right of colonial peoples to freedom and independence through assistance in raising levels of physical and mental health, the resolution appealed to the Member States of WHO to introduce or develop in their health education programmes the teaching of the principles of racial equality and non-discrimination, with a view to promoting good mental health and in recognition of the fundamental right of every human being to health. The Assembly also adopted a resolution voicing satisfaction with the speedy assistance given by WHO to the Congo and requesting the Director-General to give similar assistance to all newly independent States who become members of WHO.

Malaria Eradication.—The Assembly approved a scheme to transfer gradually, over a period of three years, the cost of field operations in the world-wide malaria eradication campaign from the special voluntary fund to WHO's regular budget. Efforts to obtain voluntary contributions to the Malaria Eradication Special Account, from governments and private sources, would continue. It was also decided that, beginning in 1961, the administrative and operational services' cost of the malaria eradication programme should be financed from WHO's regular Budget. These measures were designed to ensure that the technical achievements in malaria eradication, with which the Assembly expressed satisfaction, would not be hampered by lack of funds.

Radiation Health.—A resolution defining the role of WHO in radiation health, including the protection of mankind from ionizing radiation hazards, whatever their source, was adopted by the Assembly. The resolution noted that present scientific knowledge provided data on the harmful biological and genetic effects on human beings of massive doses of inonizing radiation and voiced recognition of the anxiety of WHO member States concerning increased exposure to ionizing radiation and approval of the action of the United Nations General Assembly to the immediate cessation of the nuclear test explosions.

Smallpox Eradication.—It was reported that the world-wide efforts to wipe out smallpox were making good progress and that most countries in which the disease was still present had now undertaken programmes to eradicate it. However, the Assembly adopted a resolution which noted that smallpox was still an important problem in international travel and that it was urgent to speed up eradication programmes.

The Assembly also gave approval to a resolution that WHO should have a flag, of the same blue colour as that of the United Nations but featuring the WHO emblem in white and gold. This emblem consists of the Aesculapian staff superimposed on the UN symbol.

The informal technical discussions this year dealt with "Recent Advances in Tuberculosis Control".

The Assembly decided that the Fifteenth Assembly should be held in Switzerland.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Discussion Week in Cancer.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Dr. R. Kaye Scott, M.D., M.S., F.R.A.C.S., F.F.R., F.C.R.A., Honorary Radiotherapist to the Austin Hospital, Melbourne, will be Guest Lecturer to the St. Vincent's Hospital, Sydney, for a discussion week in cancer, to be held from April 17 to 21, 1961, under the supervision of Dr. Leicester Atkinson. Morning demonstrations will be held in the Radiotherapy Department and afternoon lectures in the Main Lecture Hall. The programme is as follows.

Monday, April 17: 10.45 a.m., "General Radiotherapeutic Techniques", Dr. R. Kaye Scott; "Oral and Ear, Nose and Throat", chairman, Dr. H. J. Ham: 2.15 p.m., "Carcinoma of the Alveolus", Dr. R. Kaye Scott; 3.30 p.m., "Carcinoma of the Larynx", Dr. N. Newton and Dr. D. Green.

Tuesday, April 18: 9.15 a.m., "Radiotherapeutic Techniques in Skin Treatments", Dr. R. Kaye Scott; 10.45 a.m., undergraduate student case demonstration; "Radiation Damage", chairman, Dr. C. de Monchaux; 2 p.m., "Radiation Damage to the Skin", Dr. M. O'Mara and Dr. J. Cramer; 3.30 p.m., "Radiation Damage to Internal Organs", Dr. J. McRae and Dr. D. P. Ewing.

Wednesday, April 19: 9.15 a.m., "Radiotherapy Techniques—Primary Breast Lesions", Dr. R. Kaye Scott; 10.45 a.m., "Radiotherapy Techniques — Post-Operative Breasts", Dr. R. Kaye Scott; "Gynæcology", chairman, Dr. M. Britnell Fraser; 2 p.m., "Surgery as the Basis of Treatment of Carcinoma of the Cervix Uteri", Dr. M. Stening; "Radiation as the Basis of Treatment of Carcinoma of the Cervix Uteri", Dr. K. McGarrity; 3.30 p.m., "Carcinoma of the Ovary", Dr. S. Devenish Meares and Dr. R. Kaye Scott.

Thursday, April 20: 9.15 a.m., "Gynæcological Techniques—Internal Radiation", Dr. R. Kaye Scott; 10.45 a.m., "Gynæcological Techniques — External Radiation", Dr. R. Kaye Scott; "Carcinogenesis and Tumour Behaviour", chairman, Dr. I. D. Miller; 2 p.m., "Changing Concepts in Carcinogenesis", Dr. K. W. Starr; discussion, Dr. R. Kaye Scott; 3.30 p.m., demonstration and discussion of cases showing complex and bizarre behaviour.

Friday, April 21: 9.15 a.m., "Radon Implants", Dr. R. Kaye Scott; 10.45 a.m., continued—"Chemotherapy of Cancer", chairman, Dr. A. B. Lilley; 2 p.m., "Chemistry", Dr. A. Freedman; "Combinations of Surgery and Chemotherapy", Professor F. F. Rundle; 3.30 p.m., "Chemotherapy of Skin Cancer", Dr. J. C. Belisario; "Combinations of Radiotherapy and Chemotherapy", Dr. B. McEwen.

The fee for attendance is £3 3s. There is no fee for members of the annual subscription course.

Course for Part I, D.D.M.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the next Part I course for the Diploma in Dermatological Medicine will begin on May 8 and continue for a period of 12 weeks, lectures being held in the afternoons only. The fee for attendance at this course is £52 10s.

Enrolments.

Those wishing to enrol on either of the above-mentioned courses are requested to notify the Course Secretary, The Post-Graduate Committee in Medicine, Herford House, 188 Oxford Street, Paddington, New South Wales, at an early date. Telephone: FA 0671.

SURGICAL SEMINARS AT ST. VINCENT'S HOSPITAL, SYDNEY.

A CHANGE has become necessary in the date of the surgical seminar stated as having been arranged for April 3, 1961, at St. Vincent's Hospital, Sydney. As this is Easter Monday, the seminar will be held instead on Monday, April 10. The other details remain unaltered.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 25, 1961.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2(1)	1	7(5)	..	1(1)	..	..	..	11
Amœbiasis	..	..	..	..	..	..	1	..	1
Ancylostomiasis	..	..	..	..	..	..	9	..	9
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(1)	15(14)	4(2)	..	..	..	7	..	30
Diphtheria	..	..	..	1(1)	..	..	..	..	1
Dysentery (Bacillary)	..	1(1)	1(1)	..	..	..	2	..	4
Encephalitis	3(3)	..	..	..	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	155(62)	109(69)	22	26(8)	13(8)	7	2	7	341
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	2(1)	..	..	..	1	..	3
Meningococcal Infection	..	2(2)	..	..	..	..	..	..	2
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	1	1	..	3	..	..	..	..	5
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	5(3)	1	..	..	..	..	..	..	6
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	7(7)	..	1	11(10)	..	..	..	..	19
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	2	..	2(2)	..	..	1	..	1	6
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	3	..	..	..	3
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	34(22)	13(11)	9(2)	5(4)	4(2)	2(1)	1	..	68
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Royal Australasian College of Surgeons.

PRIMARY EXAMINATION FOR FELLOWSHIP.

At the primary examination for Fellowship of the Royal Australasian College of Surgeons held in Melbourne and Dunedin in March, 1961, the following candidates were approved: Graeme Sorby Adams, William Alexander Joseph Campbell, Charles Peter Clarke, Lloyd Stewart Coats, Brian Garland Dunstan Cohen, Brian Thomas Collopy, Margaret Myfanwy Davis, David John Gillett, Rodney John Hall, Alan Thomas Hosking, John Frederick Leditschke, Hans Joseph Lorbeer, Lionel Geoffrey Lukin, Ruth Valda Magnus, James Webster Mewett, Peter John Morris, Peter Graeme Petty, Peter James Phillips, John Peterson Royle, Ian Albert Shumack, David Henri Thomas.

FACULTY OF ANÆSTHETISTS.

Primary Examination for Fellowship.

At the primary examination for fellowship of the Faculty of Anæsthetists of the Royal Australasian College of Surgeons held in Melbourne and Dunedin in March, 1961, the following candidates were approved: David Cranleigh Thomson Bush, John Reed Hankey, Beryl Eileen Jack, Donald John Taylor.

Scientific Day in Sydney.

The Faculty of Anæsthetists of the Royal Australasian College of Surgeons announces that a scientific day will be held in Sydney on May 6, 1961, in the Students' Lecture Theatre at the Royal North Shore Hospital. All medical practitioners are invited to be present. The programme is as follows.

10.15 a.m., Opening address, Sir Norman Nock, Chairman, Royal North Shore Hospital; 10.30 a.m., "Radioisotopes for the Surgeon", Mr. Hedley Atkins, D.M. (Oxon), Sims Commonwealth Travelling Professor; 11.45 a.m., "Blood—Profit and Loss in Surgery", Mr. T. S. Reeve, Surgical Research Officer, Unit of Clinical Investigation, Royal North Shore Hospital; 2 p.m., "Studies on Iron Absorption", Dr. R. J. Walsh, F.R.A.C.P., Director, Red Cross Blood Transfusion Service; 2.45 p.m., "Experiments with the Heart-Lung Preparation", Professor T. Cecil Gray, M.D., Sims Commonwealth Travelling Professor.

Admission to lunch will be by ticket only. It will assist catering arrangements if tickets are obtained early by sending a cheque for £1 to the Honorary Secretary before May 4, but they will also be available on the day. The address of the Honorary Secretary is as follows: Dr. George M. Davidson, 86 Elizabeth Bay Road, Elizabeth Bay.

Notes and News.

Fourteenth International Conference on the Study of the Patient as a Person.

The fourteenth International Conference on the Study of the Patient as a Person, organized by the Bossey Group, will take place at the Getsemani Foundation, Casale Corte Cerro, Novara, Italy, from July 31 to August 5, 1961. The main theme of the conference will be the meaning of self-realization for man as a person, and the official languages will be French, German, English and Italian. Further information may be obtained from Dr. Paul Plattner, Sanatorium Wyss, Münchenbuchsee bei Bern, Switzerland.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Celermajer, John Marian, M.B., B.S., 1960 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

Vines, Arthur Peter, M.B., B.S., 1956 (Univ. Sydney), Dept. of Public Health, Okapa via Kainantu, Eastern Highlands, New Guinea.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Bird, Beverley Ann, M.B., B.S., 1960 (Univ. Sydney), District Hospital, Geelong.

Diary for the Month.

APRIL 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APRIL 5.—Western Australian Branch, B.M.A.: Branch Council.
APRIL 5.—Victorian Branch, B.M.A.: Branch Council.
APRIL 6.—South Australian Branch, B.M.A.: Council Meeting.
APRIL 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full data in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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